



CONTRACT NO. 93-323
FINAL REPORT
APRIL 1999

Reclamation of Automotive Batteries: Assessment of Health Impacts and Recycling Technology

Task 2: Assessment of Health Impacts

CALIFORNIA ENVIRONMENTAL PROTECTION AGENCY



**AIR RESOURCES BOARD
Research Division**

**RECLAMATION OF AUTOMOTIVE
BATTERIES: ASSESSMENT OF HEALTH
IMPACTS AND RECYCLING TECHNOLOGY**

**TASK 2:
ASSESSMENT OF HEALTH IMPACTS**

**FINAL REPORT
CONTRACT NO. 93-323**

PREPARED BY:

**ARCADIS GERAGHTY & MILLER
555 CLYDE AVENUE
P. O. BOX 7044
MOUNTAIN VIEW, CA 94039**

PREPARED FOR:

**CALIFORNIA AIR RESOURCES BOARD
RESEARCH DIVISION
2020 L STREET
SACRAMENTO, CA 95814**

APRIL 1999

ACKNOWLEDGMENTS

Principal Authors:

Michelle Montano
Stefan Unnasch
Pamela Franklin

Bendix Environmental Research, Inc.

Joseph M. Rut
Selina Bendix, Ph.D., R.E.A.

ARB Project Officers:

Marla Mueller - Research Division
Marijke Bekken - Mobile Source Control Division

The authors would like to thank the following staff of the Air Resources Board: Jackie Johnson, Nancy Steele, Michelle Dobbins, Kathleen Nolan and Manjit Ahuja for their assistance with this study. The authors would also like to thank Karen Randles and her associates at the Office of Environmental Health Hazard Assessment. Many individuals in industry, and private and government agencies have also provided invaluable assistance in acquiring the most up to date information on the state of battery technology and battery recycling technology.

This report was submitted in fulfillment of Task 2 of ARB Contract A93-323, "Reclamation of Automotive Batteries: Assessment of Health Impacts and Recycling Technology," by ARCADIS Geraghty & Miller under sponsorship of the California Air Resources Board. Work was completed as of October 1998.

For more information about the ARB's Research Division,
its research and activities, please visit our Web site:

<http://www.arb.ca.gov/rd/rd.htm>

Abstract

This final report for Task 2: Assessment of Health Impacts compares the relative health and hazard impacts of electric vehicle (EV) battery recycling technology. This analysis only compares batteries to other batteries, and as such does not represent an absolute impact assessment, in that the recycling processes for these batteries were not compared to other sources of environmental pollution. The purpose of Task 2 was to compare the relative impact of recycling EV batteries in terms of cancer, toxicity, and ecotoxicological potential, as well as leachability, flammability, and corrosivity/reactivity hazards. These impacts were evaluated for lead-acid, nickel-cadmium, nickel-metal hydride, sodium sulfur, sodium-nickel chloride, lithium-iron sulfide and disulfide, lithium-polymer, lithium-ion, and zinc-air batteries. Health/hazard impacts were evaluated for recycling methods, including smelting, electrowinning, and other appropriate techniques that apply to different battery technologies.

A multi-attribute impact analysis was performed on the health and hazard effects resulting from the recycling and disposal of each selected battery type. The methodology for this assessment is based largely on the protocol developed by OEHHA and ARB for prioritizing toxic air contaminants. This methodology used a semi-qualitative ranking to weight the relative impact of the different battery types and different recycling options against each other. A health and environmental impact score was developed for each battery constituent. The total amount of spent battery material consumed per mile was calculated for each battery type, based on the battery's life, weight, and composition. The fate of battery materials was then determined for different recycling processes (i.e., smelting, electrowinning) in order to estimate the amount of materials distributed to the air, water, landfills, and recycled product. Using this information, a health/hazard score was developed for each battery type.

This methodology is meant for comparison purposes only. There is a great deal of uncertainty surrounding this analysis. Battery constituents are reasonably well known, but they vary somewhat with battery manufacturer and are likely to change as battery development progresses. Recycling technologies for many of these batteries are in their infancy; in some cases, actual emissions have been measured, while, in other cases, engineering judgement has to be applied to adapt emissions factors from other presumably similar processes. In addition, there can be substantial uncertainty surrounding the health impact values (cancer potency factors, maximum contaminant levels, etc.). Because of uncertainties, interpretations made from this analysis should be viewed with caution. Based on this assessment effort, lead-acid batteries processed by smelting operations seem to have a larger potential negative health impact than the same batteries processed by other means or than other battery types. The NaNiCl_2 batteries also rank relatively more toxic. On an overall basis, the more advanced batteries, such as NiMH, lithium-polymer, and lithium-ion, represent a great improvement over conventional lead-acid battery technology, both in terms of battery performance and impacts from recycling the spent batteries.

TABLE OF CONTENTS (cont.)

3.2.5 Corrosivity (Not included in the protocol)	36
3.2.6 Reactivity.....	37
3.2.7 Leachability (Not included in the protocol)	37
SECTION 4: RESULTS AND CONCLUSIONS	38
4.1 Health/Hazard Score.....	38
4.1.1 Results	38
4.1.2 Factors that Drive the Rankings	40
4.2 Flammability/Reactivity Scores	41
4.2.1 Flammability	41
4.2.2 Reactivity	42
4.3 Uncertainty	43
4.4 Conclusions	44
REFERENCES	45
LIST OF ACRONYMS.....	48
APPENDIX A	50
APPENDIX B.....	59

TABLE OF CONTENTS

SECTION 1: INTRODUCTION	1
SECTION 2: BATTERY RECYCLING EMISSIONS	3
2.1 Battery Performance	3
2.1.1 Battery Weight	3
2.1.2 Expected Vehicle Range	6
2.1.3 Mileage per Year	6
2.1.4 Battery Life	6
2.1.5 Specific Battery Weight	7
2.2 Battery Composition	7
2.2.1 Spent Battery Material Streams	9
2.3 Fate of Battery Components	9
2.3.1 Recycling Scenario	9
2.3.2 Air Emission Factors	9
2.3.3 Dioxin Emissions	11
2.3.4 Emission Factor Considerations	11
2.3.5 Distribution of Battery Materials to Product, Land, and Water	13
2.3.6 Lead-Acid and Sealed Bipolar Lead-Acid	13
2.3.7 Nickel-Cadmium	15
2.3.8 Nickel-Metal Hydride	16
2.3.9 Sodium-Sulfur	20
2.3.10 Sodium-Nickel Chloride	20
2.3.11 Lithium Batteries	21
2.3.12 Zinc-Air	23
2.3.13 Comparison with Other Metals Recycling Processes	23
2.3.13.1 Silver Recovery from Photo Processing	24
2.3.13.2 Metal Recovery from Electronic Components	24
2.3.13.3 Industrial Metal Recycling	25
2.3.14 Environmental Loadings	25
SECTION 3: HEALTH/HAZARD IMPACT ANALYSIS	30
3.1 Health/Hazard Evaluation Methodology	30
3.2 Health/Hazard Attributes	32
3.2.1 Carcinogenic Impact	34
3.2.2 Toxicity (Noncancer)	34
3.2.3 Ecotoxicological Potential	35
3.2.4 Flammability	35
3.2.4.1 DOT Hazardous Material Regulations	35
3.2.4.2 The MSDSs and Other Sources	36
3.2.4.3 NFPA Hazardous Materials Ratings	36

LIST OF TABLES

Table 1. Batteries Under Development for Electric Vehicle Use	4
Table 2. Battery Performance Specifications.....	5
Table 3. Battery Compositions (Mass Percent)	8
Table 4. Spent Battery Material to be Recycled (g/mi)	10
Table 5. Air Emission Factors.....	13
Table 6. Fate of Battery Materials-Sealed Lead-Acid, Smelting.....	14
Table 7. Fate of Battery Materials-Lead-Acid, Smelting.....	15
Table 8. Fate of Battery Materials-Lead-Acid, Electrowinning	15
Table 9. Fate of Battery Materials-NiCd, Pyrometallurgical Process	16
Table 10. Fate of Battery Materials-NiMH AB ₂ , Chemical Process.....	17
Table 11. Fate of Battery Materials-NiMH AB ₂ , Physical/Chemical Process	17
Table 12. Fate of Battery Materials-NiMH AB ₂ , Pyrometallurgical Process	18
Table 13. Fate of Battery Materials-NiMH AB ₅ , Chemical Process.....	18
Table 14. Fate of Battery Materials-NiMH AB ₅ , Physical/Chemical Process.....	19
Table 15. Fate of Battery Materials-NiMH AB ₅ , Pyrometallurgical Process	19
Table 16. Fate of Battery Materials-Sodium-Sulfur	20
Table 17. Fate of Battery Materials-Sodium-Nickel Chloride.....	21
Table 18. Fate of Battery Materials-Lithium-Iron Sulfide.....	22
Table 19. Fate of Battery Materials-Lithium-Iron Disulfide	22
Table 20. Fate of Battery Materials-Lithium-Polymer	22
Table 21. Fate of Battery Materials-Lithium-Ion.....	22
Table 22. Fate of Battery Materials-Zinc Air	23
Table 23. Summary of EV Battery Market Share Estimates	26
Table 24. California Projected Electric Vehicle Population.....	27
Table 25. Potential Environmental Loading of Battery Materials, 2000.....	28
Table 26. Potential Environmental Loading of Battery Materials, 2010.....	29
Table 27 Health and Hazard Attributes.....	33
Table 28. Health/Hazard Attributes Included in the Protocol.....	34
Table 29. DOT Regulation Labeling Requirements	35
Table 30. Battery Constituents Score.....	39
Table 31. Health/Hazard Score	39

LIST OF FIGURES

Figure 1. Project Quantity of Spent Battery Material Generated.....	12
Figure 2. Normalized Results for Each Battery Type and Recycling Option.....	44

LIST OF TABLES (cont.)

Table 32. Weighted Flammability Potential, Including Plastic	42
Table 33. Weighted Flammability Potential, Excluding Plastic	42
Table 34. Weighted Reactivity Potential	43
Table 35. Weighted Reactivity Potential, Excluding Electrolyte	43

this study, recycling process evaluations, and emissions estimates. The hazard attributes are then related to the emissions from each material in a battery to provide a multi-attribute relative impact analysis.

Even if the recycling technologies and infrastructure discussed in Task 1 are developed, it does not necessarily follow that recycling electric vehicle batteries will resolve the related environmental issues. For example, the batteries must be transported from the consumer to the recycling facility, often with one or more intermediate steps. The recycling operations themselves produce emissions and wastes. The human health and hazard assessment will rank the battery technologies against each other, based on the ultimate fate of the battery components in the recycling process; whether in a recycled product, in wastewater, air emissions, or a landfill.

Ranking batteries and health impacts is a complex exercise. In order to compare battery types, the analysis must be normalized on a standard per-car basis, as opposed to a per-battery basis, because of the different number of batteries required depending on the battery type. In addition, even the per-car analysis must be adjusted to reflect the differing lifetimes (i.e., number of charge cycles) provided by each battery type. The constituents of each battery must be identified and tracked throughout the battery's recycling and/or disposal process, in order to account for all of the battery material. With these adjustments, the final environmental loadings are expressed as a mass-per-mile and total ton-per-year inventory, consistent with traditional vehicle emissions. Also consistent with traditional vehicle emissions, the loadings include a variety of pollutants, though some pollutants (such as cadmium) will be unusual compared to traditional vehicle emissions.

In addition to the quantitative environmental loading calculations, health and hazard implications of all battery constituents are determined in order to compare the batteries on a per mile basis. The relative importance of different health/hazard attributes is assessed so that battery rankings reflect the greater importance to human health of certain characteristics (e.g., toxic exposure from air emissions) over others (e.g., corrosivity risks during recycling). Similarly, the per-mile environmental loadings for each constituent include figures for water and land waste, in addition to air emissions, so that the relative importance of loadings to each media can be examined. An overall health/hazard assessment is based on the weighted results of the rankings in this study.

SECTION 1

INTRODUCTION

The Low Emission Vehicle and Clean Fuels program, adopted by CARB in September 1990, required the phased introduction of vehicles that meet the progressively more stringent exhaust emissions standards. One element of this program mandated that, beginning in 1998, two percent of new vehicles sold by large-volume manufacturers must be zero-emission vehicles (ZEVs). This requirement increased to a maximum of 10 percent in year 2003. In 1996, ARB modified the ZEV regulations to incorporate a market-based approach that will speed the introduction of cleaner vehicles nationwide and allow time for development of the electric vehicle (EV) infrastructure and longer-range batteries. Mandatory requirements to produce and offer specified numbers of ZEVs for sale will go into effect in 2003. However, this report is based on the CARB regulations adopted in 1990, because Task 1 was completed prior to the regulation change. This does not affect the relative rankings of the batteries, since these rankings were based on gallons per mile (g/mi) emissions from spent batteries, not on total emissions from a battery type. Since the majority of batteries contain substances that are toxic and may pose a health hazard to humans and the environment, it is important to identify these potential hazards and ensure that a robust recycling chain is established. This is needed to accommodate the increasing number of batteries, and minimize any potential health and environmental effects.

Both in California and nationally, the necessary recycling technology and infrastructure exists to accommodate the current supply of spent conventional lead-acid batteries. One facility in the United States is currently capable of recycling nickel-cadmium (NiCd) and nickel-metal hydride (NiMH) batteries. Some recycling facilities exist for other battery types.

In *Task 1: Assessment of Recycling Technology*, several different EV battery technologies were studied and ranked, based on their performance and recyclability. A review of battery technology was performed in Task 1 as a basis for determining which batteries were to be considered for a detailed relative impact analysis. The batteries chosen for further study are lead-acid, sealed bipolar lead-acid, NiCd, NiMH (AB₂ and AB₅ types), sodium-sulfur (NaS), sodium-nickel chloride (Na-NiCl₂), lithium-iron sulfide, lithium-iron disulfide, lithium-polymer, lithium-ion, and zinc-air. A general description of each battery, battery performance, and the stage of research for each battery is provided in Section 2.1 of the Task 1 report.

Task 2: Assessment of Health Impacts assesses the relative health/hazard impact of the recycling of these batteries, and provides a ranking based on each battery's projected effect on human health and the environment. It should be noted that the batteries are only ranked against each other, not against other sources of environmental pollution. As such, this report does not represent an absolute health impact assessment, but rather a relative health risk comparison. The result is a ranking of batteries, based on their hazardous characteristics, to determine which of the batteries poses the greatest health risk to humans and the environment, compared to the other batteries. This report includes a summary of the battery materials, including their toxic and hazardous components, applicable regulatory requirements, and battery performance characteristics. The assessment of battery impacts combines the fate of hazardous materials with health/hazard attributes (e.g., carcinogenicity, toxicity, leachability, flammability, reactivity). The first step is to determine the amount of hazardous substances introduced into the environment by the different types of spent batteries. The term "spent battery" means a battery that is no longer useful for providing electric power to a vehicle and thus enters the recycling stream. The fate of each battery component is determined from the recycling technologies analyzed in Task 1 of

Table 1. Batteries Under Development for Electric Vehicle Use¹

System	Specific Energy (Wh/kg) (C/3)	Peak Power (W/kg) (80% DOD)	Energy Efficiency (%)	Battery Life (cycles) (C/3 & C/2)	Self-Discharge (% per 48h)	Production Cost (US\$/kWh) ²	Operating Temperature (°C) ³	Ability to Fast Charge ³	Commercial Availability ³	Existing Recycling Facilities ³
Acidic aqueous solution										
Lead Acid	30 to 50	100 to 300	>80	500 to 1,500	0.6	70 to 150	Ambient	Yes	Now	Yes
Sealed Bipolar Lead-Acid	55	450	NA	1000+	<15	150	-30° to 65°C	Yes	NA	Yes
Zinc/bromine	70 to 80	35 to 100	75	200 to 2,000	40	100	20° to 40°C	No	No	No
Alkaline aqueous solution										
Nickel cadmium	50 to 60	130 to 235	75	2,000	2	300	Ambient	Yes	Now	Yes
Nickel-metal hydride ⁴	80-120	175	>70	800+	>10	200	-30° to 60°C	<1h	1995	Yes
Nickel-iron	40 to 60	80 to 150	60	500 to 2,000	3	150	Ambient	Yes	1993/4	Yes
Aluminum/air	220 to 300	160	<50	NA	NA	NA	NA	NA	No	No
Iron/air	100	90	60	500+	NA	50 ¹	NA	NA	No	No
Zinc/air	120 to 200	30 to 80	60	600+	NA	85	Ambient	No	No	Yes ⁶
Molten Salt										
Sodium-sulfur	90 to 120	130 to 180	85	500+	0	250	350°C	Yes	1995	No
Sodium-nickel chloride	90 to 130	60 to 130	NA	1,000+	NA	250	300°C	Yes	No	No
Lithium-iron sulfide	60 to 130	100 to 200	80	1,000+	NA	150	450°C	Yes	No	Yes ⁷
Solid State										
Lithium polymer	85 to 130	80 to 85	80 to 85	1000	0.03	100 ¹	Ambient to -120°C	NA	NA	Yes ⁷
Lithium-ion ⁸	108	216		800+	<1 ⁹	NA	Ambient to 60°C	Yes ⁵	1998	NA
USABC Goals¹⁰										
USABC mid-term	80 to 100	150 to 200	75	600	<15	150	-30° to +65°C	<6h	1995 required	Required
USABC long-term	200	400	80	1,000	<1	100	-40° to +85°C	3 to 6h	1998 required	Required

1. Reference 1

2. Reference 2

3. Reference 3.

4. Reference 4

5. Reference 5.

6. Reference 6

7. Reference 7

8. Reference 8

9. Reference 9

10. United States Advanced Battery Consortium

NA = Not Available

SECTION 2

BATTERY RECYCLING EMISSIONS

Emissions from battery recycling are determined in terms of spent battery material usage per mile and recycling facility emissions. The amount of battery material that must be recycled is determined from the battery weight, mileage accumulation, and battery life. The first part of this study determines the frequency of battery recycling and expresses the spent battery material on a per mile basis.

2.1 Battery Performance

Performance parameters of EV batteries are important in the overall hazard assessment since a battery that contains more toxic materials, such as a NiCd battery, will improve its ranking once its longer life is taken into account. Some battery performance parameters (listed in the Task 1 report and repeated here in updated version as Table 1) are used in Task 2 to determine the battery material emissions produced during the recycling process. The following discussion covers the method and inputs for determining spent battery material.

2.1.1 Battery Weight

Performance parameters are listed in Table 2. The values for battery weight in Table 2 represent the total weight of the entire battery pack in the vehicle, not the weight of the individual cells. The battery weights will represent a balance between vehicle range expectations and energy carrying limitations. For some light-duty EVs, a 500 kg battery will provide a marginal to acceptable range of 87 to 287 miles. Lithium iron-disulfide and zinc-air batteries have such high energy densities that prototype EVs have achieved over 300 miles range. We would expect that EVs built with these batteries would have a lower battery weight. For purposes of simplicity in calculation, a light-duty vehicle (LDV) battery for lead-acid through NiMH in Table 2 is assumed to weigh 500 kg, a medium duty vehicle (MDV) battery weighs 700 kg, and a heavy-duty vehicle (HDV) battery weighs 2000 kg. This is generally consistent with the literature. For batteries with a higher specific energy, including NaS through zinc-air in Table 2, the range is assumed to be limited to 200 miles per charge for an LDV battery and 110 miles per charge for an MDV battery, and the battery weight is calculated from equation 1. HDV battery weights are only considered for lead-acid batteries. MDV and HDV batteries are considered for the purpose of assessing total battery recycling demand.

$$\text{Weight} = \frac{\text{Range (mi)} \times \left(1 + \frac{\text{self-discharge}}{2}\right) \times \text{energy consumption (kWh / mi)}}{\text{specific energy (Wh / kg)} \times (1 \text{ kW} / 1000 \text{ Wh})} \quad (1)$$

2.1.2 Expected Vehicle Range

The battery weights are used to determine the anticipated vehicle range. The specific energy and self-discharge are typical values taken from the literature. The data on energy consumption per mile was not always available in the literature, but energy consumption is heavily dependent on the size of the vehicle and the way in which the vehicle is driven. Commonly accepted values are 0.2 kWh/mi for an electric LDV and 0.4 kWh/mi for an electric MDV. Data on energy consumption was difficult to find for heavy-duty electric vehicles. However, for an internal combustion vehicle (ICV), a light-duty engine consumes about one-tenth of the energy of a heavy-duty transit bus. Therefore, a value of 2 kWh/mi was taken for electric HDVs. The self-discharge factor (percent discharge per 48 hours) takes into consideration the reduction in range due to charge loss when the battery is not in use. The formula for calculating range is shown in equation 2.

$$Range (mi) = \frac{Weight (kg) \times [specific energy (Wh / kg)] \times (1 kWh / 1000 Wh)}{[energy consumption (kWh / mi)] \times \left(1 + \frac{self - discharge}{2}\right)} \quad (2)$$

2.1.3 Mileage per Year

EV mileage accumulation is used to estimate the battery life, based on cycle life testing. Annual mileage is also used to represent the spent battery material on a per mile basis. Average miles driven per year may be less in the near term, since new electric vehicle owners will likely drive their EVs on more short errand trips around town, while still using their gasoline vehicles for longer trips. However, 11,000 miles per year is a median value for LDVs, based on the EMFAC model for calculating vehicle miles traveled (VMT) [10]. First year mileage for LDVs is closer to 15,000 miles per year. Medium-duty vehicles typically travel 12,000 miles per year, and 40,000 miles per year is typical of a heavy-duty transit bus (Source ARB). Thus, these median values represent conservatively low values. It should be noted that, although the California ZEV mandate only includes light-duty vehicles, it is likely that a small number of medium and heavy-duty vehicles will also be EVs. It was necessary to assume a certain population of MDV and HDV EVs in order to determine the necessary recycling capacity for handling California's spent EV batteries. If both of these vehicle classes were ignored, the estimated capacity may be inadequate.

2.1.4 Battery Life

This study estimates the battery life in years. Ideally, battery life can be determined from field experience in EVs. Since such data are not available for all batteries, laboratory data and manufacturer estimates must be used to determine battery life. The maximum practical battery life, in years, is determined from the literature, where available. The battery life can also be predicted from the cycle life. The cycle life indicates how many times a battery can be charged and discharged. Ideally, the cycle life combined with the energy density represents the total amount of energy that can be delivered by the battery before it must be replaced. The charging profile is not expected to have an impact on battery life. Discharging a battery to 80 percent of its charge should have the same effect on battery life as discharging the same battery 20 percent, four times. This premise is used to calculate the "theoretical" battery life. For the range assumptions and battery weight assumptions shown in Table 2, the theoretical life is considerably longer than the manufacturers' estimates of calendar life, even if the battery is assumed to be driven to only 80 percent of its range, or 80 percent depth of discharge. However, some batteries have only been tested at the cell level, and have not been adequately tested at the vehicle level.

Table 2. Battery Performance Specifications

Battery Type	Battery Weight (kg)	Energy Consumption of EV (kWh/miles)	Specific Energy (Wh/kg)	Self-discharge Coefficient	Expected Range (mi/charge)	Mileage per Year ² (mi/yr)	Total Cycle Life (charges)	Max. Theoretical Life (years)	Estimated Life (years)	Specific Battery Weight (g/mi)
Pb – Acid	LDV	0.2	35	0.60%	87	11,000	600	3.81	2.00	22.73
	MDV	0.4	35	0.60%	61	12,000	600	2.44	2.00	29.17
	HDV	2	35	0.60%	35	40,000	600	0.42	0.42	25.00
Pb – Acid (Sealed Bi-Polar)	LDV	0.2	45	0.60%	112	11,000	1000	8.16	3.00	15.15
	MDV	0.4	45	0.60%	79	12,000	1000	5.23	3.00	19.44
	HDV	2	45	0.60%	45	40,000	1000	0.90	0.90	25.00
NiCd	LDV	0.2	55	2%	136	11,000	2000	19.80	7.00	6.49
	MDV	0.4	55	2%	95	12,000	2000	12.71	4.00	14.58
Ni-Metal-Hydrate AB ₂	LDV	0.2	71	10%	169	11,000	1000	12.29	6.00	7.58
	MDV	0.4	71	10%	118	12,000	1000	7.89	4.00	14.58
Ni-Metal-Hydrate AB ₃	LDV	0.2	71	10%	169	11,000	1000	12.29	6.00	7.58
	MDV	0.4	71	10%	118	12,000	1000	7.89	4.00	14.58
NaS	LDV	0.2	120	10%	200	11,000	800	11.64	6.00	5.30
	MDV	0.4	120	10%	110	12,000	800	5.87	4.00	8.02
Na-NiCl ₂	LDV	0.2	110	10%	200	11,000	1000	14.55	8.00	4.34
	MDV	0.4	110	10%	110	12,000	1000	7.33	5.00	7.00
LiFeS	LDV	0.2	95	10%	200	11,000	1000	14.55	5.00	8.04
	MDV	0.4	95	10%	110	12,000	1000	7.33	3.00	13.51
LiFeS ₂	LDV	0.2	160	10%	200	11,000	1000	14.55	5.00	4.77
	MDV	0.4	160	10%	110	12,000	1000	7.33	3.00	8.02
Li – Polymer	LDV	0.2	110	0.03%	200	11,000	1000	14.55	5.00	6.61
	MDV	0.4	110	0.03%	110	12,000	1000	7.33	3.00	11.11
Li-Ion ¹	LDV	0.2	113	1.00%	200	11,000	800	11.64	5.00	6.47
	MDV	0.4	113	1.00%	110	12,000	800	5.87	4.00	8.15
Zn – Air	LDV	0.2	150	0.00%	200	11,000	600	8.73	7.00	3.46
	MDV	0.4	150	0.00%	110	12,000	600	4.40	5.00	4.89

1. Data based on limited available information.

2. Based on EMFAC specific model year weighted average [10]

Table 3. Battery Compositions (Mass Percent)

Battery Type	Battery Material (mass fraction, %)																					
	(Data based on manufacturer MSDSs and engineering estimates.)																					
	Al	Sb	As	Cd	C, graphite	Cl	Co	Cr	Cu	F	Fe	Pb	Li	Mn	Ni	P	Na	S	Sn	Ti	V	Zn
Pb - Acid	--	2.0	0.2	--	--	--	--	--	--	--	--	60.0	--	--	--	--	--	--	0.2	--	--	--
Sealed Bipolar Pb-Acid	--	0.3	--	0.3	--	--	--	--	0.8	--	--	72.0	--	--	--	--	--	--	0.2	--	--	--
NiCd	--	--	--	18.0	--	--	0.2	--	--	--	--	--	0.03	--	25.6	--	--	--	--	--	--	--
Ni-Metal-Hydrate AB ₂	0.4	--	--	--	--	--	--	2.1	--	--	43.5	--	--	--	24.0	--	--	--	--	0.8	7.1	--
Ni-Metal-Hydrate AB ₃	0.3	--	--	--	--	--	1.7	--	--	--	43.5	--	--	0.8	29.2	--	--	--	--	--	--	--
NaS	--	--	--	--	1.3	--	--	--	--	--	--	--	--	--	--	--	8.3	15.4	--	--	--	--
Na-NiCl ₂	18.0	--	--	--	--	11.3	--	--	--	--	--	--	--	--	15.3	--	11.9	--	--	--	--	--
LiFeS	--	--	--	--	--	--	--	--	--	--	33.0	--	8.3	--	--	--	--	18.8	--	--	--	--
LiFeS ₂	--	--	--	--	--	--	--	--	--	--	22.5	--	11.3	--	--	--	--	26.3	--	--	--	--
Li - Polymer	--	--	--	--	--	--	--	--	6.0	--	--	--	15.0	--	--	--	--	--	--	--	--	--
Li-Ion ¹	--	--	--	--	18.1	--	--	--	--	3.4	--	--	2.1	--	35.4	0.9	--	--	--	--	--	--
Zn - Air	--	--	--	--	--	--	--	--	--	--	--	--	--	--	10.0	--	--	--	--	--	--	40.0

1. Data based on limited available information.

Thus, their maximum calendar life cannot be accurately determined, although researchers and developers have made estimates of their calendar lives. The high energy consumption of buses results in batteries being cycle- life limited rather than calendar-life limited.

$$\text{Max. Theoretical Life (years)} = \frac{\text{range(mi / charge)} \times 80\% \times (\text{charges / life})}{(\text{mi / yr})} \quad (3)$$

2.1.5 Specific Battery Weight

The specific weight of the battery (in g/mile) is the estimated battery weight divided by the estimated miles driven in the total life of the battery. Where possible, the actual life of the battery is used in this calculation, instead of the theoretical life. This value is then used to determine the spent battery material on a per mile basis for each battery.

$$\text{Specific Battery Weight (g / mi)} = \frac{[\text{Battery Wt. (kg)}] \times (1000 \text{ g / kg})}{(\text{mi / yr}) \times \text{Life (years)}} \quad (4)$$

2.2 Battery Composition

For the purpose of determining the fate of battery components, all compositions were reduced to the metal elemental compositions; for instance, nickel is present in the NiMH batteries as Ni(OH)₂, but the battery composition (and subsequent fate and distribution) is calculated in terms of the amount of Ni.

The composition of each battery type was determined in order to assess the amount of toxic or potentially hazardous material in each battery. Table 3 documents the battery compositions in detail. The battery compositions are taken from manufacturer MSDS reports [11], manufacturer estimates, or government studies whenever possible. In cases where these data are not available, engineering estimates are based on the battery half-cell reactions. Some batteries, such as the NiMH, vary in composition from manufacturer to manufacturer and by intended use. The compositions presented here are those determined to be appropriate for electric vehicle use.

Materials listed under the “others” category in Table 3 are non-hazardous materials, including items such as stainless steel, calcium, hydrogen, oxygen, and rare earth metals. The hydrogen and oxygen lumped into the “others” column are simply the percentage of these elements that are present in hydroxides, such as nickel hydroxide and lithium hydroxide. In cases where these substances are present in the batteries, the percentage of nickel or lithium is listed separately under the column for those specific metals. Stainless steel, although it contains chromium, is considered non-hazardous because the chromium is in the form of Cr(III), which is relatively non-hazardous, rather than Cr(VI), which is highly toxic. Stainless steel is also usually recycled. Other unspecified materials include separators, leveling agents, insulation in high temperature batteries, composites, and unspecified metals. These are unspecified in the MSDS because the manufacturer was not required to disclose them for environmental compliance, due to their apparently non-hazardous nature. An example of such a material is the alpha

Table 4. Spent Battery Material to be Recycled (g/mi)

Battery Type	Spent Battery Material (g/mi)																							Electrolyte		Casing					
	Al	Sb	As	Cd	C ₆ graphite	Cl	Co	Cr	Cu	F	Fe	Pb	Li	Mn	Ni	P	Na	S	Sn	Ti	V	Zn	Zr	Other	H ₂ SO ₄	Other	Plastic	Other			
Pb - Acid	--	0.45	0.05	--	--	--	--	--	--	--	--	13.64	--	--	--	--	--	--	0.05	--	--	--	--	--	2.32	5.09	--	1.14	--		
	MDV	--	0.58	0.06	--	--	--	--	--	--	--	17.50	--	--	--	--	--	--	0.06	--	--	--	--	--	2.98	6.53	--	1.46	--		
	HDV	--	0.50	0.05	--	--	--	--	--	--	--	15.00	--	--	--	--	--	--	0.05	--	--	--	--	--	2.55	5.60	--	1.25	--		
Pb - Acid (Sealed Bi-Polar)	LDV	--	0.05	--	0.04	--	--	--	0.11	--	--	10.91	--	--	--	--	--	--	0.03	--	--	--	--	--	--	3.11	--	0.91	--		
	MDV	--	0.06	--	0.05	--	--	--	0.15	--	--	14.00	--	--	--	--	--	--	0.04	--	--	--	--	--	--	3.99	--	1.17	--		
	HDV	--	0.08	--	0.06	--	--	--	0.19	--	--	18.00	--	--	--	--	--	--	0.05	--	--	--	--	--	--	5.13	--	1.50	--		
NiCd	LDV	--	--	1.17	--	--	0.01	--	--	--	--	--	0.00	--	1.66	--	--	--	--	--	--	--	--	--	1.06	--	1.88	0.71	--		
	MDV	--	--	2.63	--	--	0.03	--	--	--	--	--	0.00	--	3.73	--	--	--	--	--	--	--	--	--	2.38	--	4.22	1.59	--		
Ni-Metal-Hydride AB ₂	LDV	0.03	--	--	--	--	--	0.16	--	--	3.30	--	--	--	1.82	--	--	--	--	0.06	0.54	--	0.19	0.42	--	0.68	0.38	--	--		
	MDV	0.05	--	--	--	--	--	0.31	--	--	6.34	--	--	--	3.50	--	--	--	--	0.12	1.04	--	0.36	0.82	--	1.31	0.73	--	--		
Ni-Metal-Hydride AB ₃	LDV	0.02	--	--	--	--	0.13	--	--	--	3.30	--	--	0.06	2.21	--	--	--	--	--	--	--	--	--	0.80	--	0.68	0.38	--	--	
	MDV	0.04	--	--	--	--	0.24	--	--	--	6.34	--	--	0.11	4.26	--	--	--	--	--	--	--	--	--	1.54	--	1.31	0.73	--	--	
NaS	LDV	--	--	--	0.07	--	--	--	--	--	--	--	--	--	--	--	0.44	0.82	--	--	--	--	--	--	1.33	--	2.12	--	0.53	--	
	MDV	--	--	--	0.10	--	--	--	--	--	--	--	--	--	--	--	0.67	1.24	--	--	--	--	--	--	2.01	--	3.21	--	0.80	--	
Na-NiCl ₂	LDV	0.78	--	--	--	0.49	--	--	--	--	--	--	--	--	0.66	--	0.52	--	--	--	--	--	--	--	--	--	--	--	1.89	--	
	MDV	1.26	--	--	--	0.79	--	--	--	--	--	--	--	--	1.07	--	0.83	--	--	--	--	--	--	--	--	--	--	--	3.05	--	
LiFeS	LDV	--	--	--	--	--	--	--	--	--	2.65	--	0.66	--	--	--	--	1.51	--	--	--	--	--	--	--	--	2.01	1.21	--	--	--
	MDV	--	--	--	--	--	--	--	--	--	4.46	--	1.11	--	--	--	2.53	--	--	--	--	--	--	--	--	--	3.38	2.03	--	--	--
LiFeS ₂	LDV	--	--	--	--	--	--	--	--	--	1.07	--	0.54	--	--	--	1.25	--	--	--	--	--	--	--	--	--	1.19	0.72	--	--	--
	MDV	--	--	--	--	--	--	--	--	--	1.80	--	0.90	--	--	--	2.11	--	--	--	--	--	--	--	--	--	2.01	1.20	--	--	--
Li - Polymer	LDV	--	--	--	--	--	--	--	0.40	--	--	--	0.99	--	--	--	--	--	--	--	--	--	--	--	2.38	--	1.98	0.86	--	--	--
	MDV	--	--	--	--	--	--	--	0.67	--	--	--	1.67	--	--	--	--	--	--	--	--	--	--	--	4.00	--	3.33	1.44	--	--	--
Li-Ion	LDV	--	--	--	--	1.17	--	--	--	0.22	--	--	0.14	--	2.29	0.06	--	--	--	--	--	--	--	--	1.25	--	0.39	0.96	--	--	--
	MDV	--	--	--	--	1.48	--	--	--	0.28	--	--	0.17	--	2.88	0.08	--	--	--	--	--	--	--	--	1.57	--	0.49	1.21	--	--	--
Zn - Air	LDV	--	--	--	--	--	--	--	--	--	--	--	--	--	0.35	--	--	--	--	--	--	--	1.39	0.35	--	1.04	0.35	--	--	--	--
	MDV	--	--	--	--	--	--	--	--	--	--	--	--	--	0.49	--	--	--	--	--	--	1.96	0.49	--	1.47	0.49	--	--	--	--	--

and beta alumina electrolyte used in NaS batteries, which is a ceramic composite. While some manufacturers include non-hazardous materials on the MSDS, many elect not to disclose these for proprietary reasons.

2.2.1 Spent Battery Material Streams

The gram-per-mile fluxes of the spent battery materials, presented in Table 4, are calculated by multiplying the specific weight in grams-per-mile times the percent of each respective material in the battery. It is assumed that the percentage of the materials entering the recycling stream is proportional to the percentage of materials in the battery. These fluxes represent those materials that enter the recycling process as part of the spent batteries. Much of these materials will ultimately be reused as new product. The small remainder is discharged to the environment (air, water, or land).

$$\text{Spent Battery Material} \left(\frac{g}{mi} \right) = \left[\text{specific battery weight} \left(\frac{g}{mi} \right) \right] \times (\% \text{ material}) \quad (5)$$

Figure 1 provides a graphic representation of the projected quantity of spent battery material that will be generated each year for each battery type.

2.3 Fate of Battery Components

2.3.1 Recycling Scenario

The most likely recycling scenario for each battery type is used to determine the fate of the battery components. In some cases, more than one recycling scenario is evaluated. Information about the recycling process was obtained through government-sponsored studies, from the recyclers, and from literature.

2.3.2 Air Emission Factors

Explicit ARB emissions factors are used for recycling processes wherever possible. Sources of other emissions factors include AP-42 and the U.S. EPA Factor Information Retrieval System (FIRE) database, which is available on-line [12]. The FIRE system contains emissions factors from AP-42, state emissions testing, and other sources. These emissions factors are characterized by pollutant, process, source classification code, and several other categories. The quality of the emissions factors in AP-42 and in the FIRE database varies considerably. These figures represent an industry-wide average for a specific process under stated conditions, and actual emissions may differ from these values. Also, AP-42 emissions factors are based on national figures, where the required emissions controls may be far more lenient than in California. Acurex Environmental has not conducted any in-house source testing of smelting or battery recycling operations. We have conducted source testing of electroplating operations, but unfortunately these data are based on hours of operation rather than mass throughputs.

Some of the available emissions factors (from smelting operations, for instance) estimate emissions of total particulate matter. In this case, the particulate emissions are assumed to be 100 percent metal, with a composition proportional to the battery composition.

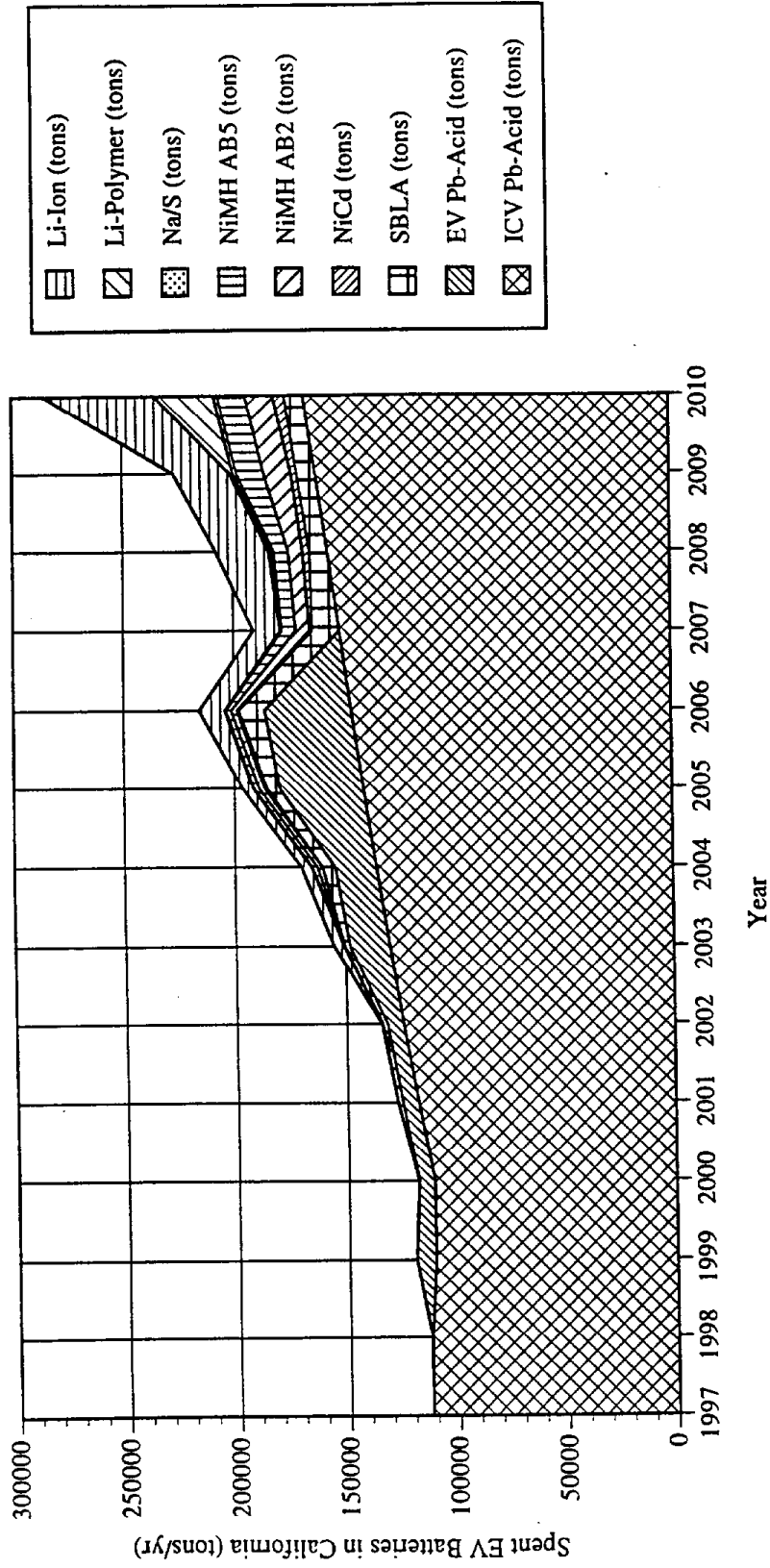


Figure 1. Level of Market Saturation from California Spent EV Batteries

In most cases, emissions are assumed to be controlled by conventional technology. For instance, in processes where baghouses or wet scrubbers are normally used, the efficiency of the control device is factored into the emissions estimate. In some cases, the given emissions factor in AP-42 or FIRE includes the control device. In other instances, the typical efficiency of a representative control device was used to calculate the controlled emissions by using this efficiency in conjunction with an emissions factor for an uncontrolled source.

In many instances, there are no explicit air emissions factors available. In these cases, engineering judgment is used to apply an emissions factor for a similar process in order to estimate emissions. For instance, there are no specific emissions factors for electrowinning lead-acid batteries. However, electrowinning is a process similar to electroplating, and emissions factors for electroplating can be used to estimate the electrowinning emissions. The air emissions factor for electroplating in the FIRE database is 0.0033 percent, which is larger than the ARB emissions factor for secondary lead smelters. The FIRE number is large because it is likely based on older facilities that only have emissions controls with about 95 percent efficiency. New electroplating facilities in California have fugitive emissions controls. Assuming control efficiency of 99.9 percent, the air emissions factor for the entire electrowinning process corresponds to approximately 0.000066 percent. Similarly, for certain processes an emissions factor for only one specific metal is available. Engineering judgment is then used to extrapolate this emissions factor to other metals undergoing the same process.

2.3.3 Dioxin Emissions

Dioxin is of concern as a possible byproduct of battery recycling by incineration of the plastic battery casings. Throughout this analysis, it is assumed that 100 percent of the plastic battery casing (polyethylene, polypropylene, Teflon, etc.) is recycled, since that is the practice in California. While incineration of plastics is not done in California, it may be practiced in other parts of the country, at secondary lead smelters or municipal waste incinerators. This report recommends that plastic battery casings not be incinerated due to the potential to produce dioxin emissions.

2.3.4 Emission Factor Considerations

The emissions from recycling facilities are a critical element of the life cycle battery emissions. Emissions are usually measured in terms of g/day from a facility, and are expressed as emissions factors in percentage of facility output. Emissions factors are much more variable than battery performance parameters because different facilities will have different degrees of emissions control. Different emissions control requirements will depend on the location of the facility and date of construction. We estimated emissions factors from various sources. The assumptions and regulations that apply to these facilities may not always be consistent; however, they are the best estimate of the case studies considered. Table 5 summarizes the key emissions factor assumptions for toxic metal air emissions. In most instances, the same emissions factors were used for other metal emissions from the same case study facilities. For example, copper emissions from assumed to be the same as lead emissions from electrowinning (0.000066 percent).

Air emissions for lead in conventional lead acid battery recycling (through smelting) are obtained from ARB for secondary lead production operations involving lead smelting. It should be noted that this factor represents more stringent controls than are used at secondary lead smelters in other parts of the country. Particulate emissions from the recycling process are assumed to have the same composition as the battery (i.e., the weight percent of the particulate matter emissions is assumed to correspond to the battery metal composition). Emissions are always assumed to be controlled. H₂SO₄ emissions are only released as fugitive mist emissions during battery breaking and hammermill crushing. There is no explicit emissions factor for H₂SO₄ in AP-42 or other sources, so a conservative estimate of 0.0001 percent is used.

No air emissions factors were found for electrowinning from AP-42, the FIRE database, or other sources. However, because the electrowinning process is very similar to electroplating, cadmium plating emissions factors (obtained from the FIRE database) are used to approximate metal emissions from electrowinning, assuming more stringent fugitive emissions controls for electrowinning than are assumed in the FIRE database. Assuming that the FIRE emissions factor of 0.0033 percent only incorporates 95 percent control efficiency, which corresponds to 5 percent release, and assuming that new facilities will have at least 99.9 percent control, which corresponds to 0.1 percent release, the ratio of 0.1 percent to 5 percent times the old emissions factor yields the new emissions factor used in this study, 0.000066 percent. In this case, the emissions factor is applied to each specific metal in Pb-acid batteries. Based on a study by Arthur D. Little of a similar chemical process (used to recycle NiMH batteries), sponsored by the National Renewable Energy Laboratory (NREL), 92 percent product recovery of metals is assumed, except for lead, for which a 98 percent recovery is assumed. Plastics are assumed to be 100 percent recovered, and the H₂SO₄ that does not escape as fugitive emissions (during battery breaking) is assumed to enter the wastewater stream.

Emissions are estimated from the recycling of two different types of lead acid batteries, regular and sealed. Representative compositions are obtained from MSDSs. No information was obtained regarding tubular lead acid or advanced lead acid battery compositions or recycling. Table 6, Table 7, and Table 8 summarize the emissions estimate for recycling lead-acid batteries.

Table 6. Fate of Battery Materials-Sealed Lead-Acid, Smelting

Chemical	Battery Composition (wt %)	Distribution of Chemicals during Recycling (wt % of total)			
		Product	Water	Land	Air
Pb	72.00%	95.00%	0.00%	5.00%	0.002%
Cu	0.75%	0.75%	0.00%	99.25%	0.003%
Cd	0.25%	0.25%	0.00%	99.75%	0.001%
Sb	0.30%	0.30%	0.00%	99.70%	0.001%
Sn	0.20%	0.00%	0.00%	100%	0.001%
Polypropylene	3.50%	100%	0.00%	0.00%	0.00%
H ₂ SO ₄	20.50%	0.00%	100%	0.00%	0.0001%
Polyethylene	2.50%	100%	0.00%	0.00%	0.00%
Total	100%	74.41%	20.50%	5.09%	0.0015%

Table 5. Air Emission Factors

Battery Material/ Process	Recycling Case	Air Emissions (% of output)	Source
Pb Smelting	Pb Acid	0.002	ARB
Pb Electrowinning	Pb Acid	0.000066	calculated
Ni Pyrometallurgical	NiCd, plastic	0.0006	Geomet
Cd Pyrometallurgical	NiCd, plastic	0.0006	Geomet
Ni Chemical	NiMH AB5	0.000066	calculated
Zn Smelting	Zn Air	.00064	ARB

2.3.5 Distribution of Battery Materials to Product, Land, and Water:

Mass balances are used to estimate the partitioning of battery components into different media through recycling, when detailed data are not available. Some of the spent materials are recycled into new products. Others are in liquid form when the recycling process is complete and are diverted into wastewater treatment plants, which remove most of the metals and neutralize any acids or bases before final discharge. Land discharge is estimated to be fifty times the water discharge. This is based on the premise that most metals should be removed from water by the wastewater treatment plant, and recovered, or converted to a sludge or filter cake, and landfilled. This emissions and discharge summary takes into account only the composition of the battery itself. Except for the production of dioxins from the incineration of plastics, it does not account for additional pollutants/materials introduced or criteria pollutants (such as NO_x, SO_x, CO, etc.). It also does not account for the energy of production of these materials or the energy needed to recycle. In other words, this analysis is a rough estimation of the distribution of the battery materials during the recycling process, in order to estimate the environmental impact of those battery components; it is not a life cycle analysis.

In the tables that follow, true zeroes are denoted by a zero without decimal places. Zeroes with two decimal places indicate that essentially no material is distributed to that media.

2.3.6 Lead-Acid and Sealed Bi-Polar Lead-Acid

An EPA study, *Characterization of Priority Pollutants from a Secondary Lead and Manufacturing Facility* (1979) [13], was referenced to determine the apportioning of metals in the various waste streams from recycling lead-acid batteries. This study characterizes the amount of material recovered and remaining in the wastewater and landfill streams, accounting for amount of pollutants removed in the treatment plant. Because this study deals with several different operations, including battery manufacturing and lead oxide manufacturing, that are not of direct interest to the recycling process of concern in this study, only the applicable waste streams are considered. The quantity of batteries recycled is used to normalize the metals in the various streams, based on the battery throughput. In many cases, the solid waste fraction of a given metal is determined by a mass balance. Because this study involved a more complex process than simple battery recycling alone, it accounts for emissions and discharges of trace amounts of certain metals that may not be listed as components in the MSDS for the two battery types considered in this evaluation. In the case of copper, the percentage recovered as product is very low (<1%), since the amount of Cu in the battery is very small and is largely incorporated in the slag that ends up as solid waste.

Table 12. Fate of Battery Materials-NiMH AB₂, Pyrometallurgical Process

Chemical	Battery Composition (wt %)	Distribution of Chemicals during Recycling (wt % of total)			
		Product	Water	Land	Air
Ni	24.01%	98.00%	0.04%	1.96%	0.0006%
Fe	43.50%	96.00%	0.08%	3.92%	0.0006%
V	7.11%	98.00%	0.04%	1.96%	0.0006%
Zr	2.50%	98.00%	0.04%	1.96%	0.0006%
Ti	0.79%	98.00%	0.04%	1.96%	0.0006%
Cr	2.14%	98.00%	0.04%	1.96%	0.0006%
Al	0.37%	98.00%	0.04%	1.96%	0.0006%
KOH	3.00%	0.00%	100%	0.00%	0.0006%
H ₂ O	6.00%	0.00%	100%	0.00%	0.00%
Leveling agents	1.00%	0.00%	100%	0.00%	0.00%
Polypropylene	5.00%	100%	0.00%	0.00%	0.00%
Other	4.58%	0.00%	100%	0.00%	0.00%
Total	100%	82.94%	14.63%	2.43%	0.0005%

Table 13. Fate of Battery Materials-NiMH AB₂, Chemical Process

Chemical	Battery Composition (wt %)	Distribution of Chemicals during Recycling (wt % of total)			
		Product	Water	Land	Air
Ni	28.41%	92.50%	0.00%	7.50%	0.000066%
Fe	43.50%	92.70%	0.00%	7.31%	0.000066%
La	3.20%	0.00%	0.00%	100%	0.000066%
Ce	0.20%	0.00%	0.00%	100%	0.000066%
Pr	1.40%	0.00%	0.00%	100%	0.000066%
Nd	0.16%	0.00%	0.00%	100%	0.000066%
Co	1.67%	83.83%	0.00%	16.17%	0.000066%
Mn	0.78%	0.00%	0.00%	100%	0.000066%
Al	0.29%	0.00%	0.00%	100%	0.000066%
KOH	3.00%	0.00%	100%	0.00%	0.00%
H ₂ O	6.00%	0.00%	100%	0.00%	0.00%
Leveling agents	1.00%	0.00%	100%	0.00%	0.00%
Polypropylene	5.00%	100%	0.00%	0.00%	0.00%
Ni(OH) ₂	5.39%	0.00%	100%	0.00%	0.00%
Total	100%	73.00%	15.39%	11.61%	0.0001%

Table 7. Fate of Battery Materials-Lead-Acid, Smelting

Chemical	Battery Composition (wt %)	Distribution of Chemicals during Recycling (wt % of total)			
		Product	Water	Land	Air
Pb	60.00%	95.00%	0.00%	5.00%	0.002%
Sb	2.00%	0.30%	0.004%	99.69%	0.001%
As	0.20%	0.00%	0.00%	100.00%	0.001%
Ca	0.20%	0.00%	0.00%	100.00%	0.001%
Sn	0.20%	0.00%	0.00%	100.00%	0.001%
Polypropylene, etc.	5.00%	99.98%	0.00%	0.00%	0.018%
H ₂ SO ₄	22.40%	0.00%	100%	0.00%	0.0001%
glass reinforced PE	10.00%	99.96%	0.00%	0.00%	0.04%
Total	100%	72.00%	22.40%	5.59%	0.0061%

Table 8. Fate of Battery Materials-Lead-Acid, Electrowinning

Chemical	Battery Composition (wt %)	Distribution of Chemicals during Recycling (wt % of total)			
		Product	Water	Land	Air
Pb	60.00%	98.00%	0.04%	1.96%	0.000066%
Sb	2.00%	92.00%	0.16%	7.84%	0.000066%
As	0.20%	0.00%	2.00%	98.00%	0.000066%
Ca	0.20%	92.00%	0.16%	7.84%	0.000066%
Sn	0.20%	92.00%	0.16%	7.84%	0.000066%
Polypropylene, etc.	5.00%	100.00%	0.00%	0.00%	0.00%
H ₂ SO ₄	22.40%	0.00%	100%	0.00%	0.0001%
glass reinforced PE	10.00%	100.00%	0.00%	0.00%	0.00%
Total	100%	76.01%	22.43%	1.56%	0.00006%

2.3.7 Nickel-Cadmium

The NiCd battery compositions are obtained from a study by Geomet Technologies, Inc., sponsored by the Electric Power Research Institute (EPRI) [14]. The process employed by INMETCO, a NiCd recycler in Pennsylvania, is used here as a representative model for NiCd battery recycling [15]. In this pyrometallurgical process, the metals are sent through both a rotary hearth furnace and an electric arc furnace to recover the Ni. Conventional control devices are assumed. The dust from the electric arc baghouse and the filter cake from the post-wet-scrubber wastewater treatment plant are collected and sent offsite for hydrometallurgical recovery of Cd. The hydrometallurgical process is assumed to have minimal air emissions. The emissions from these two processes are added to obtain a total emissions rate. The Geomet study has calculated the amount of metal in the wastewater, solid waste, and air emissions streams. The amount of product is then estimated, using a mass balance. Table 9 lists the assumed emissions rates for NiCd recycling.

2.3.9 Sodium-Sulfur

The composition of NaS batteries is estimated, in part, from information obtained from Silent Power, a company which has developed a recycling process for these batteries [17]. The amounts of Na, S, and stainless steel were obtained from this source. MSDS information is available only for the individual constituents of the battery, not for the overall battery composition. Therefore, the weight percentages of alpha alumina, beta alumina ($\text{Na}_2\text{O} \cdot x\text{Al}_2\text{O}_3$, where x = an integer from 5 to 11), insulation, the cell case, and carbon fiber/graphite are estimates.

Although there are several different recycling processes for NaS batteries, the recycling process developed by Silent Power is used as the basis for determining the fate and distribution of the battery materials. The Silent Power process involves acidic oxidation followed by the Claus process, yielding sodium sulfate and elemental sulfur.

Air emissions from this process are negligible, with the exception of small amounts of H_2SO_4 . Product recoveries for Na, S, and steel were estimated by Silent Power to be 90 percent. The recovery of alpha and beta alumina is assumed to equal 90 percent as well. The insulation, cell case, and carbon fiber/graphite are assumed to be landfilled. Of the materials that were not recovered, the remaining distribution is assumed to be apportioned 98 percent as non-hazardous solid waste and 2 percent as wastewater. Distribution of emissions from this process is listed in Table 16.

Table 16. Fate of Battery Materials-Sodium-Sulfur

Chemical	Battery Composition (wt %)	Distribution of Chemicals during Recycling (wt % of total)			
		Product	Water	Land	Air
Na	8.3%	90.00%	0.20%	9.80%	0.0001%
S, as H_2SO_4	15.4%	90.00%	0.20%	9.80%	0.0001%
Stainless steel	20.0%	90.00%	0.20%	9.80%	0.00%
Alpha alumina	20.0%	90.00%	0.20%	9.80%	0.00%
Beta alumina	20.0%	90.00%	0.20%	9.80%	0.00%
Insulation	5.0%	0.00%	0.00%	100%	0.00%
Cell case	10.0%	0.00%	0.00%	100%	0.00%
Carbon fiber/graphite	1.3%	0.00%	0.00%	100%	0.00%
Total	100%	75.33%	0.17%	24.50%	0.00002%

2.3.10 Sodium-Nickel Chloride

The MSDSs for Na- NiCl_2 batteries do not provide quantifications of the overall battery composition, although they list the following battery contents: NaCl, Na metal, nickel, and nickel chloride. The battery composition is therefore estimated, using the stoichiometric half-cell reaction. Steel is assumed to be the only casing material.

A process for recycling Na- NiCl_2 batteries has been developed at the bench scale by AEG Corporation [18]. This process involves leaching and precipitation followed by metallurgical processing of nickel and steel and is used as the basis for Na- NiCl_2 recycling in this study. AEG estimates that 90 percent of the nickel may be recovered in this process. The air emissions for nickel are the same as those applied to NiCd and NiMH smelting, which were adapted from the Geomet study [14]. A control device with an efficiency of 95 percent is assumed for this evaluation. This emissions factor has been adapted

Table 14. Fate of Battery Materials-NiMH AB₅, Physical/Chemical Process

Chemical	Battery Composition (wt %)	Distribution of Chemicals during Recycling (wt % of total)			
		Product	Water	Land	Air
Ni	28.41%	94.72%	0.00%	5.28%	0.000066%
Fe	43.50%	99.66%	0.00%	0.34%	0.000066%
La	3.20%	100%	0.00%	0.00%	0.000066%
Ce	0.20%	100%	0.00%	0.00%	0.000066%
Pr	1.40%	100%	0.00%	0.00%	0.000066%
Nd	0.16%	100%	0.00%	0.00%	0.000066%
Co	1.67%	100%	0.00%	0.00%	0.000066%
Mn	0.78%	100%	0.00%	0.00%	0.000066%
Al	0.29%	100%	0.00%	0.00%	0.000066%
KOH	3.00%	0.00%	100%	0.00%	0.000%
H ₂ O	6.00%	0.00%	100%	0.00%	0.000%
Leveling agents	1.00%	0.00%	100%	0.00%	0.000%
Polypropylene	5.00%	100%	0.00%	0.00%	0.000%
Other	5.39%	0.00%	100%	0.00%	0.000%
Total	100%	82.96%	15.39%	1.65%	0.0001%

Table 15. Fate of Battery Materials-NiMH AB₅, Pyrometallurgical Process

Chemical	Battery Composition (wt %)	Distribution of Chemicals during Recycling (wt % of total)			
		Product	Water	Land	Air
Ni	28.41%	99.997%	0.002%	0.003%	0.0006%
Fe	43.50%	99.266%	0.733%	0.00%	0.0006%
La	3.20%	99.999%	0.00%	0.00%	0.0006%
Ce	0.20%	99.999%	0.00%	0.00%	0.0006%
Pr	1.40%	99.999%	0.00%	0.00%	0.0006%
Nd	0.16%	99.999%	0.00%	0.00%	0.0006%
Co	1.67%	98.503%	0.00%	1.496%	0.0006%
Mn	0.78%	99.999%	0.00%	0.00%	0.0006%
Al	0.29%	99.999%	0.00%	0.00%	0.0006%
KOH	3.00%	100%	0.00%	0.00%	0.0006%
H ₂ O	6.00%	0.00%	100%	0.00%	0.000%
Leveling agents	1.00%	0.00%	100%	0.00%	0.000%
Polypropylene	5.00%	100%	0.00%	0.00%	0.000%
Other	5.39%	0.00%	100%	0.00%	0.000%
Total	100%	87.26%	12.71%	0.03%	0.0005%

Table 18. Fate of Battery Materials-Lithium-Iron Sulfide

Chemical	Battery Composition (wt %)	Distribution of Chemicals during Recycling (wt % of total)			
		Product	Water	Land	Air
Li	8.3%	65.00%	0.00%	35.00%	0.0001%
Fe	33.0%	65.00%	0.00%	35.00%	0.0001%
S	18.8%	65.00%	0.00%	35.00%	0.0001%
Electrolyte	25.0%	65.00%	0.00%	35.00%	0.0001%
Casing (Teflon)	15.0%	100%	0.00%	0.00%	0.00%
Total	100%	70.25%	0.00%	29.75%	0.0001%

Table 19. Fate of Battery Materials-Lithium-Iron Disulfide

Chemical	Battery Composition (wt %)	Distribution of Chemicals during Recycling (wt % of total)			
		Product	Water	Land	Air
Li	11.3%	65.00%	0.00%	35.00%	0.0001%
Fe	22.5%	65.00%	0.00%	35.00%	0.0001%
S	26.3%	65.00%	0.00%	35.00%	0.0001%
Electrolyte	25.0%	65.00%	0.00%	35.00%	0.0001%
Casing (Teflon)	15.0%	100%	0.00%	0.00%	0.00%
Total	100%	70.25%	0.00%	29.75%	0.0001%

Table 20. Fate of Battery Materials-Lithium Polymer

Chemical	Battery Composition (wt %)	Distribution of Chemicals during Recycling (wt % of total)			
		Product	Water	Land	Air
Li	15.0%	65.0%	0.0%	35.0%	0.0001%
Polymer	30.0%	10.0%	0.0%	90.0%	0.0001%
Polyethylene	13.0%	65.0%	0.0%	35.0%	0.0001%
Cu	6.0%	65.0%	0.0%	35.0%	0.0001%
Metal, unspecified	6.0%	65.0%	0.0%	35.0%	0.0001%
Composite	30.0%	10.0%	0.0%	90.0%	0.0%
Total	100%	32.0%	0.0%	68.0%	0.0001%

Table 21. Fate of Battery Materials-Lithium Ion

Chemical	Battery Composition (wt %)	Distribution of Chemicals during Recycling (wt % of total)			
		Product	Water	Land	Air
Li	2.1%	65.00%	0.00%	35.00%	0.0001%
Ni	35.4%	65.00%	0.00%	35.00%	0.0001%
P	0.9%	65.00%	0.00%	35.00%	0.0001%
F	3.4%	65.00%	0.00%	35.00%	0.0001%
C	18.1%	65.00%	0.00%	35.00%	0.0001%
O	19.3%	65.00%	0.00%	35.00%	0.0001%
Organic electrolyte	6.0%	10.00%	0.00%	90.00%	0.0001%
Teflon	14.9%	100%	0.00%	0.00%	0.00%
Total	100%	66.97%	0.00%	33.16%	0.0001%

for Fe and Al emissions as well. A 90 percent product recovery rate is assumed for Na, Al, Fe, and steel. The remainder of these metals is assumed to be distributed to the land and water in a 98 percent: 2 percent ratio. The process emissions rates are shown in Table 17.

Table 17. Fate of Battery Materials-Sodium-Nickel Chloride

Chemical	Battery Composition (wt %)	Distribution of Chemicals during Recycling (wt % of total)			
		Product	Water	Land	Air
Na	11.9%	90.00%	0.20%	9.80%	0.0001%
Ni	15.3%	90.00%	0.20%	9.80%	0.0006%
Cl	11.3%	0.00%	100%	0.00%	0.0001%
Al	18.0%	90.00%	0.20%	9.75%	0.05%
Steel	43.5%	90.00%	0.20%	9.80%	0.00%
Total	100%	79.83%	11.48%	8.68%	0.009%

2.3.11 Lithium Batteries

There are four types of lithium batteries under consideration in this study: LiFeS, LiFeS₂, Li-polymer, and Li-ion. The compositions of the LiFeS and the LiFeS₂ batteries are estimated using the stoichiometric half-cell reactions as a basis, combined with engineering estimates. The Li-polymer battery composition is based on semiquantitative information from the *Journal of Power Sources* 43-44 (1993), 195-208 [19]. Li-ion battery composition is based on an article by G. Sarre et al. (SAFT) and input from EPRI battery specialists combined with engineering estimates [8]. Current versions of lithium-ion batteries have a relatively high weight percentage of nickel, which is present on the positive electrode in a molar ratio of approximately 1 mole Li: 2 moles Ni. Advanced versions of this battery are likely to replace most of the nickel with manganese, although some nickel may still be present in a molar ratio approximating 1 mole Li: 1.8 moles Mn: 0.2 moles Ni. The recycling process used by Toxco, a U.S. company with a recycling facility in British Columbia, salvages 10 to 65 percent of the inputs as products, while 35 percent of the mass input is emitted as off-gases or non-hazardous solid waste [7]. For this analysis, 65 percent of all metals, 10 percent of polymer or composite materials, and 100 percent of Teflon/casing material are assumed to be recycled. The remainder is assumed to be landfilled. No hazardous air emissions or wastewater are generated from this process. Emissions of metals (Li and Fe) into the air are assumed to consist only of random fugitive losses of about 0.0001 percent. The distribution of battery materials to air, water, land, and recycled product for each of the lithium batteries is shown in Tables 18-21. The lithium that ultimately goes to the landfills is in an oxidized, non-hazardous state.

2.3.13.1 Silver Recovery from Photo Processing

The majority of spent photo processing chemicals and wash waters are discharged to sanitary sewers. However, before they can be discharged, they must meet very strict discharge limits, and the chemicals are often regenerated several times before they are officially declared spent. Silver is the primary contaminant that is recovered as silver halides, from the emulsion on the film and paper. There are several processes for recovering silver and regenerating the chemicals. These include metal replacement cartridges (MRC), electrowinning, and ion exchange, by various combinations of these three methods, depending on which chemical solution is being desilvered/regenerated [20].

Metal Replacement Cartridges MRC units are typically constructed of sealed plastic housing, with capacities ranging from 0.75 gal to 15 gal. The center of the cartridge contains the metal replacement material, usually steel wool, although iron, aluminum, and zinc mesh screens are also used. As the silver-containing solution (developer, bleach, fixer, wash, or stabilizer) flows through the unit, the active metal in the center (iron) reacts with the silver thiosulfate and goes into solution, while the silver settles out as a sludge. MRCs can recover as much as 80 percent of the silver in complex as metallic silver. The effluent can contain as little as 1 mg/L. However, when the MRC is at about 85 percent capacity, the efficiency drops off and breakthrough occurs, resulting in effluents containing as much as 50 to 100 mg/L. When this occurs, the MRC should be replaced [20].

Electrowinning Electrolytic recovery is a very efficient method of silver recovery. The process operates by passing an electric current from the anode to the cathode, through the concentrated silver solution. The silver plates out as nearly pure metal on the cathode. Typically, a current of 8 amperes can provide a recovery of 31 g/hr, as long as the silver concentration in the solution does not drop below 1 mg/L. Below this concentration, the efficiency deteriorates quickly. The most efficient silver recovery method is electrowinning, followed by a metal replacement cartridge. This configuration can recover as much as 99 percent of the silver in solution [20].

Ion Exchange Ion exchange is an effective silver recovery method for solutions with a low silver concentration, such as wash waters. However, it is usually only feasible for photoshops that have large volumes of dilute wash waters or strict effluent discharge limits. In wash waters, silver is present in a complex with thiosulfate ions. This complex must be removed by an anion-exchange resin. The resin can remove as much as 96 percent of the silver complex into a much more concentrated solution. The concentrated solution is then treated with electrowinning to remove the silver in metallic form [20].

2.3.13.2 Metal Recovery from Electronic Components

Metal recovery associated with printed circuit boards is often limited to recovery from the metallic plating or rinse solutions associated with the manufacture of the printed circuit boards, rather than the recycling of the boards themselves. The plating bath chemicals may include nickel, copper, tin, lead, and gold. The spent plating solutions containing metals are often recovered using electrolyte reactors, which yield high recoveries (about 90 percent for Cu; not as high for tin and lead). In other cases, metal concentrates may be sent to smelters or other secondary recovery operations. Electrowinning may be used to recover metal hydroxide sludge from the filter press cakes of acid stream precipitators [21].

Some electronic components used in commercial applications are completely recycled. Components such as computers and monitors can be disassembled and valuable components, such as

2.3.12 Zinc-Air

The recycling process for zinc-air batteries used as the basis for this evaluation is the one used by Recovery and Reclamation, a Texas-based company [6]. In this process, the batteries are broken open and the cells are washed with KOH to flush off the ZnO. The ZnO/KOH solution is run through a filter press to remove the KOH. The dried ZnO is either sold to a zinc company or a plating company. The air emissions from recovery of metallic zinc through smelting was estimated using emissions rates for zinc smelting processes in California. A product recovery of 90 percent is assumed. The remaining metal is assumed to be apportioned to solid waste and wastewater in a 98 percent: 2 percent ratio. The distribution of emissions from this process is listed in Table 22. Here again, for the nickel smelting, the same air emissions factor is applied here as was applied to NiCd and NiMH smelting operations, developed from the Geomet study [14].

Table 22. Fate of Battery Materials-Zinc Air

Chemical	Battery Composition (wt %)	Distribution of Chemicals during Recycling (wt % of total)			
		Product	Water	Land	Air
Zn	40.0%	90.00%	9.81%	0.19%	0.00064%
Ni	10.0%	90.00%	9.80%	0.20%	0.00060%
KOH	30.0%	0.00%	100%	0.00%	0.00%
Polypropylene, PE	10.0%	100%	0.00%	0.00%	0.00%
Teflon	3.0%	100%	0.00%	0.00%	0.00%
Lamp black carbon	2.0%	0.00%	0.00%	100%	0.00%
Catalysts (metals)	0.5%	90.00%	9.81%	0.19%	0.00064%
Other	4.5%	0.00%	0.00%	100%	0.00%
Total	100%	58.45%	34.95%	6.60%	0.0003%

2.3.13 Comparison with Other Metals Recycling Processes

Recycling practices vary considerably for metals used in non-EV battery applications. Recycling is fairly common for commercial uses of toxic metals, while many consumer products are disposed of in landfills. Examples of hazardous metals that are disposed of by consumers include the following:

- Consumer batteries (which include NiMH and Li-ion)
- Photographic negatives
- Consumer electronics, components, circuit boards
- Mirrors
- Brass pens
- Copper wire, heat exchangers

Municipalities provide little opportunities to support the recycling of consumer products. Presumably, several pounds of metals per person, including copper, lead from solder, silver from photographic products, and others, find their way into landfills per person. Private individuals are exempt from many of the hazardous waste rules that businesses must abide by.

Many toxic metals are recycled by businesses. These include photographic silver, electronic components, batteries, automotive parts, and others.

Table 23. Summary of EV Battery Market Share Estimates

Battery	Estimated market potential		Estimated market share of new LDVs & MDVs (%)		
	Maximum long term market	Comment	Near term 2000	Mid term 2005	Long term 2010
Lead-Acid	100	Current market share.	38	0	0
Sealed Bipolar Lead-Acid	70	Now in pilot production.	15	10	6
NiCd	40	Cadmium Council estimate, expensive.	5	0	0
NiMH-AB ₂	30	Performs as well as NiCd but less toxic. Pilot plant being built.	10	10	7
NiMH-AB ₃	30	Performs as well as NiCd but less toxic. Pilot plant being built.	10	10	7
NaS	50	Supported by Ford.	2	0	0
Na-NiCl ₂	50	Built by AEG for Mercedes.	0	0	0
LiFeS	20	Long term substitute for Na-NiCl ₂ corrosion problems	0	0	0
LiFeS ₂	30	Disulfide has higher energy, corrosion problems.	0	0	0
Zn/Air	10	Primary battery, not rechargeable.	0	0	0
Li Polymer	50	Promising long-term battery, some current military applications.	0	30	50
Li Ion	50	Low cost, high energy. Currently used in motorcycles and consumer products.	20	40	30
New Light and Medium Duty EVs in CA			37,595	203,350	217,440

memory chips and microprocessors, are removed. Other components, such as capacitors, coils, and resistors, can be desoldered and reused, but generally are not reused in the United States. Batteries are removed for recycling and the computer is sent to a shredder. The shredded material is smelted to recover metals, including lead, copper, and gold. Other electronic items, such as cash registers, are recycled in a similar manner [21].

2.3.13.3 Industrial Metal Recycling

Many metals used in commercial applications are recycled. Toxic metals that are frequently recycled include lead from bearings and copper wire. Nickel and chromium are present in stainless steel, which is extensively recycled. This mixed metal mixed is called mischmetal and is a primary constituent for new stainless steel production.

2.3.14 Environmental Loadings

The total environmental loading associated with each battery type is calculated according to Eq. 6 below. This equation represents the total environmental loading from electric vehicles sold in any given year, including the recycled products, and air, water, and land emissions of each component. The ton-per-year emissions take into account the anticipated market share of each battery and the total vehicle population. The market share and total vehicle population were presented in Tables 4 and A2 of *Task 1: Assessment of Recycling Technology*, and are repeated here as Table 23 and Table 24 (total EVs for a given year for LDVs, MDVs and HDVs) respectively. The (mi/yr) is also given in Table 24 as average annual mileage and (g material/mi) is given for each spent battery material for each battery type in Table 4. The environmental loadings presented in Table 25 and Table 26 represent the total mass emissions of each material in the spent batteries that will ultimately enter the waste stream, based on the total number of spent batteries available. Note that there will be a delay due to the lag in recycling. This delay varies depending on the cycle life of each battery type. Therefore, the figures in Tables 25 and 26 represent the maximum potential impact of recycling all EV batteries present in 2000 and 2010.

$$\text{Spent Battery Material (tons/yr)} = (1.02 \times 10^{-6} \text{ tons/g}) \times (\text{g material/mi}) \times (\text{mi/yr}) \times (\% \text{ market share}) \times (\# \text{ vehicles}) \quad (6)$$

Assuming that this entire amount then enters the recycling stream, a percentage of this amount will become product, and additional percentages will enter the air, water, and land media, as indicated in Tables 6-22. Thus, the environmental loadings represent the sum of the potential recycled product, air, water, and land emissions.

Table 25. Potential Environmental Loading of Spent Battery Materials, 2000

Battery Type		Spent Battery Material to be Recycled from EVs Sold in 2000																						
		Al	Sb	As	Cd	C	Cl	Co	Cr	Cu	F	Fe	Pb	Li	Mn	Ni	P	Na	S	Sn	Ti	V	Zn	Zr
PbA	LDV		88.7	9.9									2690							9.9				
	MDV		4.4	0.5									132							0.5				
SBLA	LDV		3.9		3.1					8.6			849							2.3				
	MDV		0.2		0.1					0.4			42							0.1				
NiCd	LDV				30.4			0.3						0.0		43.1								
	MDV				2.6			0.0						0.0		3.7								
NiMH AB ₂	LDV	1.6							8.3			171				94.5					3.1	28.0		9.9
	MDV	0.1							0.6			12.5				6.9					0.2	2.1		0.7
NiMH AB ₃	LDV	1.0						6.7				171			3.1	115								
	MDV	0.1						0.5				12.5			0.2	8.4								
NaS	LDV					0.7												4.6	8.5					
	MDV					0.0												0.3	0.5					
NaNiCl ₂	LDV	0					0										0	0						
	MDV	0					0										0	0						
LiFeS	LDV											0		0						0				
	MDV											0		0						0				
LiFeS ₂	LDV											0		0						0				
	MDV											0		0						0				
Li-polymer	LDV													0										
	MDV													0										
Li-Ion	LDV					121					22.8			14.5		238	6.2							
	MDV					5.9					1.1			0.7		11.4	0.3							0
Zn-Air	LDV															0								0
	MDV															0								0

Table 24. California Projected Electric Vehicle Population

Year	New Vehicles ¹	Percent LDV/ EVs ¹	New LDV EVs ¹	Accumulated LDV EVs ¹	Retired LDV EVs ¹	Total LDV EVs ¹	New MDV EVs ²	Retired MDV EVs	Total MDV EVs	New HDV EVs	Retired HDV EVs	Total HDV EVs	LDV EVs	MDV EVs	HDV EVs
1997	1,722,632	1%	17,226	17,226	0	17,226	200	0	200	20	0	20	45	1	2
1998	1,751,977	2%	35,040	52,266	0	52,266	1,080	0	1,280	20	0	40	136	7	3
1999	1,781,311	2%	35,626	87,892	332	87,560	1,439	0	2,720	20	0	60	228	14	5
2000	1,810,738	2%	36,215	124,107	946	123,161	1,380	0	4,100	50	0	110	320	21	9
2001	1,837,506	5%	91,875	215,982	2,109	213,873	2,520	0	6,620	50	0	160	556	34	13
2002	1,863,129	5%	93,156	309,139	4,083	305,055	3,600	0	10,220	50	0	210	793	53	17
2003	1,890,059	10%	189,006	498,144	9,201	488,944	6,300	0	16,520	50	0	260	1,271	86	21
2004	1,917,075	10%	191,708	689,852	16,353	673,499	8,939	0	25,459	50	0	310	1,751	132	25
2005	1,944,701	10%	194,470	884,322	31,533	852,790	8,880	0	34,339	50	0	360	2,217	179	29
2006	1,974,344	10%	197,434	1,081,756	51,877	1,029,880	8,820	0	43,159	50	20	390	2,678	224	31
2007	2,004,244	10%	200,424	1,282,181	78,086	1,204,095	8,761	200	51,720	50	20	420	3,131	269	34
2008	2,034,347	10%	203,435	1,485,616	109,474	1,376,142	8,520	1,080	59,159	50	20	450	3,578	308	36
2009	2,063,716	10%	206,372	1,691,987	144,245	1,547,742	8,400	1,439	66,120	50	50	450	4,024	344	36
2010	2,093,395	10%	209,340	1,901,327	181,542	1,719,784	8,100	1,380	72,839	50	50	450	4,471	379	36
2011	2,123,640	10%	212,364	2,113,691	219,620	1,894,071	7,739	2,520	78,059	50	50	450	4,925	406	36
2012	2,154,028	10%	215,403	2,329,093	256,869	2,072,224	7,739	3,600	82,198	50	50	450	5,388	427	36
2013	2,184,133	10%	218,413	2,547,507	290,632	2,256,874	7,739	6,300	83,638	50	50	450	5,868	435	36
2014	2,216,788	10%	221,679	2,769,186	319,086	2,450,100	7,739	8,939	82,438	50	50	450	6,370	429	36
2015	2,243,271	10%	224,327	2,993,513	343,659	2,649,854	7,739	8,880	81,296	50	50	450	6,890	423	36

Average annual mileage	LDV	MDV	HDV
Average energy consumption (kWh/mi)	13,000	13,000	40,000
	0.20	0.40	2.00

1. Reference 22

2. Reference 23

SECTION 3

HEALTH/HAZARD IMPACT ANALYSIS

A multi-attribute impact analysis was performed on the health and hazard effects resulting from the recycling and disposal of each selected battery type. Several obstacles prevent a rigorous quantitative analysis of the integrated risks of battery recycling. Performing a risk assessment of cancer cases, toxic exposures, and recycling hazards would require extensive evaluations of emissions sources, population distributions, atmospheric conditions, and other appropriate parameters, which are outside the scope of this study. Instead, this study uses a semi-qualitative ranking to weight the relative impact of different battery types and different recycling options against each other. Appendix A provides the assessment methodology used to determine the likely health and hazard impacts from each battery type and recycling approach. Although there is a separate discussion of the hazard issue included in this section (addressing flammability, reactivity, leachability, and corrosivity), the hazard impact has been integrated into one health/hazard score, to allow evaluation of all the battery types with a single parameter. This chapter describes the methodology used and the justifications employed. The impact analysis begins with the transport of the spent batteries from the collection center to the recycling facility. It does not consider in-use hazards or vehicle operational hazards.

3.1 Health/Hazard Evaluation Methodology

In performing a health and hazard impact assessment, it is necessary to translate the emissions of various battery materials of concern into a health or hazard impact. There are both quantitative and qualitative methodologies to perform this task. Quantitatively, impact assessment methodologies have been developed that utilize available health or hazard information to quantify impacts. A typical quantitative impact assessment might take the stack emissions rate from a given battery recycling operation and apply a dispersion model to the emissions, to determine the concentration of pollutants in the air that reaches the target population. Using the concentration of the pollutant in the air (and sometimes land and water), and using risk factors developed by scientists and public health agencies, the assessor determines the likelihood that exposure to that pollutant at that given concentration for a given number of years will significantly impact the health of the target population, and by how great a degree. These methodologies use factors such as cancer potency and reference exposure limits (RELs)¹, below which adverse health/hazard impacts are not anticipated. One comprehensive methodology that has been developed for lead impacts is the Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK). It quantifies lead intake from air, water, soil, dust, food, paint, and other sources, and translates this intake into blood lead levels for children up to 7 years old, using pharmacokinetic modeling. The IEUBK model was designed to predict exposure and total uptake of lead only. Since there are many metals and other potentially hazardous substances under consideration in this project, the IEUBK model could not be utilized for the relative assessment under development here.

Sometimes it is not possible to perform a quantitative impact assessment because of questionable emissions data quality, unknown meteorology impacts, insufficient knowledge about health endpoints for the pollutant(s) under consideration, and other factors. Such a situation arose in this research effort. In this health/hazard impact assessment, we found uncertainty surrounding the quantification of the emissions rates themselves. Many of the batteries evaluated are not yet in commercial production, and, therefore, are of somewhat uncertain final formulation, and the recycling procedures may not yet be fully developed for each particular battery. Emissions from recycling batteries at existing facilities were not

¹ These and subsequent health impact terms are defined in the footnotes to Table 27.

Table 26

		Spent Battery Material to be Recycled from EVs Sold in 2010																						
Battery Type		Al	Sb	As	Cd	C	Cl	Co	Cr	Cu	F	Fe	Pb	Li	Mn	Ni	P	Na	S	Sn	Ti	V	Zn	Zr
PbA	LDV		0	0									0								57			
	MDV		0	0									0								2.6			
SBLA	LDV		9		7.2					19.8			1964								13.5			
	MDV		0.4		0.3					1.0			97.5								0.7			
NiCd	LDV				0			0							0		0							
	MDV				0			0							0		0							
NiMH AB ₂	LDV	6.3							33.6			693										18	162	57
	MDV	0.4							2.5			51.5				382						1.4	12.1	42
NiMH AB ₃	LDV	4.2						27.3				693			12.6	464								
	MDV	0.3						2.0				51.5			0.9	42.7								
NaS	LDV						0												0	49.2				
	MDV						0												0	2.9				
NaNiCl ₂	LDV	0					0										0		0					
	MDV	0					0										0		0					
LiFeS	LDV														0					0				
	MDV													0						0				
LiFeS ₂	LDV														0					0				
	MDV														0					0				
Li-polymer	LDV														0					0				
	MDV														0									
Li-Ion	LDV					1053					198													
	MDV				51.5						9.8			126	5.9	2061	54							
Zn-Air	LDV															100	2.8						0	
	MDV															0							0	

ten times that of another battery does not necessarily have ten times the impact. For ease in examination, the results were normalized.

Emissions to the air were given prime consideration, while emissions to the water and discharges to the land were not specifically incorporated. The environmental exposures resulting from the battery recycling processes are mostly from air, and inhalation of airborne contaminants is the primary exposure route for potential receptors. It is very difficult to evaluate ingestion exposure pathways, given the limited emissions data. In addition, evaluation of these secondary pathways is beyond the scope of this study. However, the effects of soil deposition of airborne metals and contaminants should not be dismissed. Over time, airborne metals and other potentially hazardous substances may deposit, accumulate, and persist in the soils near recycling facilities. This could pose health concerns to individuals living in the vicinity of the facility from exposure to contaminated soil and windblown dust, long after the facility has shut down. In fact, health effects associated with exposure to lead, originating from vehicular or stationary sources, have been attributed to inadvertent ingestion of lead-contaminated soils, especially by children.

3.2 Health/Hazard Attributes

Health and hazard attributes considered in this study include carcinogenicity, chronic and acute toxicity, ecotoxicological potential, flammability, and reactivity, as shown in Table 28. The weightings attempt to reflect the relative importance of each attribute, as well as, to some extent, the population exposed. For example, flammability and reactivity will mostly affect the worker, whereas airborne carcinogens will impact the surrounding community as a whole. Given the relatively low emissions rates, health impacts from chronic exposure were given significant weights. Each battery constituent was evaluated for each of these attributes. The resultant scores were summed and multiplied by the g/mi spent battery mass of that compound and the percent of that compound emitted to the air was associated with the given recycling technology, as shown in Equation 7. Finally the results were normalized. Table 27 summarizes health and hazard attributes for the various battery materials. Further health and environmental information is available in the references, as well as in Appendix B.

well quantified, leading to excessive uncertainty in a quantitative analysis. Although the metals under analysis were relatively well studied in terms of human health effects, data gaps still exist. The general tendency in most analyses is to assume zero impact where there are no data to suggest otherwise. This may or may not be an appropriate assumption for compounds that have not yet been thoroughly evaluated. Finally, during the course of this project, the ARB identified inorganic and compound lead as a Toxic Air Contaminant (TAC), with no identified threshold REL. Without an identified REL for lead, it is not possible to estimate health impacts by using quantitative impact assessment procedures. Therefore, we decided to utilize a qualitative impact assessment methodology for this project.

There are a variety of qualitative assessment methodologies available that could have been utilized in this project (e.g., Graedel, Allenby, Steele [33]). For this study, we decided to modify the Toxic Air Contaminant Prioritization Scheme, developed by the Office of Environmental Health Hazard Assessment (OEHHA) and the ARB. The methodology is simple to understand. It utilizes the health information that would normally be used in a quantitative impact assessment, where available, such as cancer potency slopes, and RELs, but also includes measures for compounds that lack these data or are less well understood. The procedure results in rankings: for example, cancer potencies are assigned a score depending on where the unit risk factor for a given compound or element falls in an established range. The individual assessment items are multiplied by the compound's emissions. Those with a high total score are good candidates for further investigation as potential contaminants of concern, under the OEHHA evaluation.

For this project, the protocol was modified to more closely reflect the project's needs. For instance, the original protocol included an item to assess the likelihood of the compound under consideration to bioaccumulate, biopersist, or biomagnify. Some organic compounds will break down over time, while metals tend to persist and might accumulate in the soil, water, and local wildlife, remaining for years after exposure has otherwise ceased. Since almost all the pollutants being investigated in this project are metals, the item offered little to the final analysis. Instead, an alternate measure of ecotoxicological potential was developed by staff at OEHHA that more closely reflects differences in the ecotoxicological potential of the pollutants under consideration in this study. The modified protocol is included in this report as Appendix A. Each component of the protocol is discussed in the following pages.

Health and environmental data, such as cancer potency factors and acute and chronic RELs, were obtained from the ARB, OEHHA, IRIS, and other public sources, and are presented in Table 27. In some cases, the impact of the pollutant is form-dependent. For example, while hexavalent chromium is highly toxic and carcinogenic, on soil deposition it rapidly binds to organic compounds, and is converted to trivalent chromium. Trivalent chromium has low toxicity and is not carcinogenic. Where the emitted form was known, the specific impacts were incorporated. The impact of all the individual compounds emitted for a given battery type and battery recycling process were then summarized, as shown in the following equation:

$$\sum (\text{battery constituent score})_i (\text{g/mi})_i (\% \text{ emitted to air})_i \quad (7)$$

where "i" is each constituent of the battery

This calculation is made for each battery type under each identified recycling option. When analyzing these totals, it is important to recall that the numbers reflect relative rankings; the scores themselves hold little meaning in terms of quantifying hazard or health impact. A battery with a score

Material	IARC/EPA Classification ^a	Cancer Potency (unit risk, m ³ /ug) ^b	Ingestion Cancer Potential ^c	RI (µda)	Flammability Rate (NFPA) ^j	Reactivity Rate (NFPA)
Al	-	-		50	1	1
Sb (SbO ₃)	2B	-	Maybe	0	0	1
As	A	3.3e-3	Yes	0	0	1
Cd	1	4.2e-3	No	3	0	0
C, black (extracts)	2B	1.7e-5 [*]	Prob. no	3 ²	0	0
Cl	-	-		7	0	0
Cr(III)	3	-	Maybe	50	0	0
Co	2B	-	Prob. no	50	0	0
Cu	3	-		2	0	0
F	3 (fluorides)	-		20	0	4
Fe	NR	-	-	50	0	0
Pb	2B	1.2e-5	Yes	Nth	0	0
Li	NR	-	-	2 ²	2	2
Mn	D	-		0	0	0
Ni	A	2.6e-4	Prob. no	0	4	2
P	D	-		0	3	2
Na (NaOH)	NR	-	-	4	1	0
S	NR	-	-	50	0	0
Sn	NR			20	0	0
Ti	NR			-	0	0
V	NR	-	-	50	0	0
Zn	D	-	-	3	0	0
Zr	NR			50	0	0
Rare Earths	NR				0	2
H ₂ SO ₄	-	-	-	10	0	1
KOH	NR			20	0	1
NaAlCl ₄	NR				3	
Plastics	NR					

* These are secondary MCLs, based on consumer acceptance levels (

+ Carbon black is listed in CAPCOA guidelines as 1% PAHs. This is NR – no ratings found

^a See end of Appendix A for a definition of these classifications.

^b The Cancer Potency or slope factor is a plausible upper-bound estimate to a potential carcinogen. These values were primarily obtained from ARB's Stationary Source Division.

^c This assesses whether ingesting the compound could potentially lead

^d The Reference Exposure Limit, or REL, is the airborne concentration

^e MCL and MCLG, are the Maximum Contaminant Level and MCLG, respectively. MCLs are enforceable standards which must be met by all public drinking water systems to which they apply. The MCLG is a health-based goal. MCLGs are non-enforceable health goals and are strictly health-based. The values were obtained from OEHHA.

^f The number of organ systems affected, and the nature of these effects

^g DHS adopted the US EPA action level under the Lead and Copper

^h The PEL, or Permissible Exposure Limit, is the maximum permitted

ⁱ The Ambient Water Quality Chronic Criteria for fresh or marine aquatic life. The values are hardness-dependent. "****" indicates that they are Suter II rather than A

^j The NOAEL, or No Observed Adverse Effect Level, is an exposure level below which no adverse effects are expected. The values were obtained from OEHHA.

^k The National Fire Prevention Association (NFPA) rates the flammability

(MCLGs); the number of organ systems affected by the compound; and the nature of those effects. Occupational PELs (Permissible Exposure Limits) are also included. PELs largely serve as a check for compounds with little general population-based data, but with potential impacts on the more heavily exposed worker population.

3.2.3 Ecotoxicological Potential (Appendix A: Items 9a and 9b)

The assessment methodology includes an item to address ecotoxicological potential. The ecotoxicological potential reflects how a compound will affect other living organisms in the environment, either directly (such as through toxicity) or indirectly through habitat alteration, such as the eutrophication of a lake resulting from excessive phosphorus input. In this assessment, ecotoxicological potential is primarily evaluated in terms of chronic toxicity to aquatic organisms. Toxicity is based on the US EPA's Ambient Water Quality Chronic Criteria for fresh or marine aquatic life (AWQC) or Suter Tier II standards (Tier II Freshwater Chronic Values, reported by Suter (1996); developed using US EPA's "Water Quality Guidance for the Great Lakes System: Proposed Rules"), and chronic toxicity to avian and mammalian wildlife, based on the lowest No Observed Adverse Effect Levels (NOAELs).

3.2.4 Flammability (Appendix A: Item 10)

Flammability is primarily a concern for spent battery collection, transportation, and dismantling, and as such, is mostly of importance to those working in these areas rather than to the general population, although certain events, such as a fire at the recycling plant, could have a substantial population impact. There are several ways to assess battery flammability. The US Department of Transportation (DOT) Hazardous Material Regulations include flammability components and the battery's Material Safety Data Sheet (MSDS) may include some flammability information. In addition, there are specific flammability and reactivity ratings developed by the National Fire Prevention Association (NFPA), which may or may not be specifically included on the MSDSs. The subsections below discuss these options for evaluating flammability.

3.2.4.1. DOT Hazardous Material Regulations

The DOT Hazardous Material Regulations govern the packaging, labeling, and acceptability of materials for transportation. These regulations indicate the degree of hazard posed by a battery during transportation for recycling, based on the hazard properties of the entire battery. Table 29 presents the DOT regulation labeling requirements for the batteries under consideration in this study. As can be seen in the table, these labels are difficult to rank, being largely descriptive in nature. For instance, is a "hazardous material" worse than a "corrosive material"? Similarly, the numeral assigned to the hazard class has no intrinsic rank meaning.

Table 29. DOT Regulation Labeling Requirements

Battery Type	DOT Regulations ¹	
	Hazard Class	Labels Required
Sealed bipolar PbA, PbA, NiCd, Zn-Air, NiMH AB ₂ , NiMH AB ₅	8	Corrosive material
NaS, Na-NiCl ₂	4.3	Dangerous when wet
LiFeS, LiFeS ₂ , Li-Polymer, Li-Ion*	9	Miscellaneous hazardous material

¹Source: 49 CFR §§ 172, 173.

Table 28. Health/Hazard Attributes Included in the Protocol

Health/Hazard Attribute	Points
Carcinogenic Potential	10
Cancer Potency (inhalation)	10
Cancer Potential (ingestion)	10
Noncancer Impacts (inhalation)	10
Noncancer Impacts (ingestion)	10
Number of Known Organ Systems Affected	5
Chronic/Acute/Reproductive or Developmental Toxicity	5
Occupational Exposure Limits	5
Ecotoxicological Potential	10
Flammability/Reactivity	5
Maximum Possible Points	80

3.2.1 Carcinogenic Impact (Appendix A: Evaluation Items 1-3)

Assessing and ranking the carcinogenic potency for each substance associated with the recycling and disposal of each battery type is based on the carcinogenic potential classifications developed by IARC and/or the US EPA, cancer potency estimates for inhalation obtained from OEHHA, and a qualitative assessment of cancer risk from ingestion as obtained from the scientific literature. The IARC and/or the US EPA recognize arsenic, chromium (VI), nickel, and certain nickel compounds as known human carcinogens. Other compounds such as antimony, cadmium, cobalt, and lead are classified as possible or probable carcinogens by IARC and the US EPA. OEHHA has developed potency slope factors for some possible and probable human carcinogens, as well as for the "known" human carcinogens.

The IARC also recognizes sulfuric acid as a known human carcinogen, but only under certain conditions that would not be encountered by the general population. IARC bases this classification on workplace studies that associate strong sulfuric acid mist with upper respiratory cancer. The only time workers in lead-acid battery recycling facilities would be exposed to sulfuric acid is during the battery breaking and wastewater neutralization processes. Arcadis Geraghty & Miller Environmental scientists have visited the GNB lead-acid recycling facility in Los Angeles and have observed the battery breaking process, but did not detect any such sulfuric acid mists. However, this does not mean that these mists never occur. Neutralized sulfuric acid in wastewater is not expected to create the exposure conditions that have been associated with excess cancer in workers, as a result of exposure to strong acid mists. For the purposes of this study, which explores the impact on the population as a whole, sulfuric acid is presented as having no carcinogenic effect.

3.2.2 Toxicity (Noncancer) (Appendix A: Items 4-8)

Toxicity is evaluated in terms of chronic, acute, reproductive, and developmental non-cancer toxicity. Chronic and acute toxicity are based on the OEHHA chronic and acute non-cancer toxicity RELs, where available; Maximum Contaminant Levels (MCLs) or Maximum Contaminant Level Goals

different from the federal regulations. It is difficult to classify any of the battery materials as corrosive (with the possible exception of electrolytes) since there is no way of knowing what the concentrations of each material might be at the time of exposure (in order to determine pH level). Such an attempt would require highly inaccurate speculation on the dilution rates in each media and how long exposure occurred after the material entered the media. Therefore, corrosivity was not further addressed in the analysis.

3.2.6 Reactivity (Appendix A: Item 10)

A reactive waste is defined (40 CFR 261.23) as a waste that exhibits one or more of the following qualities: (1) it is normally unstable at ambient conditions, undergoing violent changes; (2) it reacts violently in water; (3) it creates a potentially explosive solution with water; (4) it generates toxic fumes when mixed with water; (5) it contains cyanide or sulfide and, when exposed to mixtures with a pH between 2 and 12.5, generates toxic fumes; (6) it is capable of exploding if exposed to a heat source; (7) it is capable of exploding at ambient conditions; or (8) it is an illegal explosive. Some battery metals, such as lithium, sodium, and sulfur, and some electrolytes can easily fall into these categories. In addition to its flammability ratings, NFPA also maintains reactivity ratings defined as follows: (4) materials that, in themselves, are capable of explosion or reaction at ambient temperatures and pressures; (3) materials that, in themselves, are capable of explosive reaction in the presence of a strong initiator, or react explosively with water, or must be heated in confinement; (2) materials that, in themselves are unstable, undergo violent chemical change, react violently with water, or form potentially explosive solutions with water; (1) materials that are normally stable but can become unstable at elevated temperatures or release some energy when reacted with water; and (0) materials that are stable and do not react with water.

Reactivity is included in the protocol, but since this information may be of interest separately, the results of the reactivity analysis are presented in Section 4.2.

3.2.7 Leachability (not included in the protocol)

Health and environmental impacts of leachability are subsumed within the ranking protocol. The principal constituents of concern for leachability of battery wastes are inorganic metals. A US EPA test procedure known as the toxicity characteristic leaching procedure (TCLP) is used to determine the leachability of materials (e.g., metals) from a waste; wastes that leach toxic metals or organic compounds at or above specified concentrations are considered hazardous by the US EPA. California uses different tests than the US EPA to determine the total and soluble concentrations of toxic and bioaccumulative substances in hazardous wastes. The regulated limits are listed as total threshold limit and soluble threshold limit concentrations [29]. These tests are used as an indication of the amount of toxic metals that could leach out under conditions similar to those found in a landfill. Leachability poses a threat to the water supply as well as to the food chain. Metals that have leached from a landfill into the groundwater can enter the food chain through agricultural water use. Once in the food chain, the concentrations of the metals can increase as they proceed higher up the food chain, a process known as biomagnification. It is difficult to evaluate leachability impacts for a generic facility, as in the case when determining emissions for battery recycling where those technologies have not yet been developed. Further, leaching is only of concern because of health endpoints of the leached material. Thus, the impacts from leaching are already incorporated into the analysis.

SECTION 4

RESULTS AND CONCLUSIONS

The scope of this study was to develop a relative comparison of the potential impacts to human health caused by the recycling of various types of spent electric vehicle batteries. The analysis presented in this section represents the impacts of one battery type compared to those of another battery type. The following twelve batteries were evaluated: lead-acid, sealed bipolar lead-acid, NiCd, NiMH (AB₂ and AB₅ types), NaS, sodium-nickel chloride (Na-NiCl₂), lithium-iron sulfide, lithium-ion disulfide, lithium-polymer, lithium-ion, and zinc-air. As indicated in Section 3, the methodology for this assessment is based largely on the protocol developed by OEHHA and ARB for prioritizing toxic air contaminants. The assessment methodology is a semi-quantitative analysis of the relative impact posed by the batteries, and is included as Appendix A.

4.1 Health/Hazard Score

A health and environmental impact score was developed for each battery constituent, shown in Table 30. The overall health/hazard score is dominated by the health impact components. Out of 80 possible points, 65 are for human health endpoints. Based on the assessment protocol used, antimony, arsenic, cadmium, lead, and nickel have the most potential negative impact, with iron, sulfur, tin, titanium, zirconium, and the electrolyte and casing materials being relatively benign. The score for each constituent of the battery was then multiplied by the emissions of that constituent to the air (battery constituent score * g/mi for spent battery pack * % emitted to air) and summed for each battery type and recycling process, as shown in Section 3.1, Equation 7.

Using this process, a battery constituent with a high score, such as cadmium, will have a large potential *negative* impact if moderate to high amounts are emitted, whereas a battery constituent that is relatively innocuous, such as sulfur, could also have a large potential impact if its emissions rate is high. Thus, a battery's relatively high overall health/hazard score may be a function of the emissions of many low- to moderate-impact constituents, or it could be a function of a single high-impact constituent emitted at a lower rate.

4.1.1 Results

The results of this summation process are presented for each battery type and recycling process in Table 31. Since the numbers in and of themselves hold no meaning, the table presents the scores in a log-normalized fashion (log [total score for that battery type and recycling option/lowest total score for any battery type]), such that a battery type/recycling process combination with a score of 0 would be anticipated to have the least anticipated health and hazard impact. Where the result is driven by particular battery constituent (i.e., most of the total impact comes from that constituent(s) with other battery components playing a much more minor role), that is noted as well. The results are also graphically presented in Figure 2.

Table 30. Battery Constituents Scores

Constituent	Score	Constituent	Score	Constituent	Score
Arsenic	65	Chromium	30	Tin	13
Cadmium	57	Lithium	25	Sulfuric acid	11
Lead	56	Chlorine	23	Sulfur	9
Antimony	51	Sodium	23	Iron	8
Nickel	45	Fluorine	22	Zirconium	7
Cobalt	35	Zinc	21	KOH	5
Manganese	33	Aluminum	20	Titanium	4
Phosphorus	33	Carbon black	20	Plastic	3
Copper	31	Vanadium	18		

Table 31. Health/Hazard Score

Battery Type	Total Score	Score Drivers	Normalized Log Score*
PbA-Smelting	1.62×10^{-2}	Lead	2.93
SBLA-Smelting	1.24×10^{-2}	Lead	2.81
Na-NiCl ₂	8.00×10^{-3}	Aluminum	2.62
NiCd-Pyromet.	9.51×10^{-4}	Nickel, Cadmium	1.70
NiMH, AB ₅ -pyromet.	8.17×10^{-4}	Nickel, Iron	1.63
NiMH, AB ₂ -Pyromet.	7.70×10^{-4}	Nickel, Iron	1.60
PbA-electrow.	5.78×10^{-4}	Lead	1.48
Zinc-Air	2.81×10^{-4}	Zinc, Nickel	1.17
Li-Ion	1.37×10^{-4}	Nickel	0.85
NiMH, AB ₅ -Chem	8.76×10^{-5}	Nickel, Iron	0.66
NiMH, AB ₅ -Phys/chem	8.76×10^{-5}	Nickel, Iron	0.66
NiMH, AB ₂ -chem	8.25×10^{-5}	Nickel, Iron	0.63
NiMH, AB ₂ -phys/chem	8.25×10^{-5}	Nickel, Iron	0.63
Li-FeS	5.13×10^{-5}	-	0.43
Li-Polymer	3.97×10^{-5}	-	0.32
Li-FeS ₂	3.33×10^{-5}	-	0.24
Na-S	1.91×10^{-5}	-	0

* A lower score means less anticipated impacts from this battery technology relative to others, using the recycling methodology specified.

The results show the greatest potential health and environmental impact, given the assumptions made for this study, for light-duty electric vehicles powered by lead acid batteries that are recycled by smelting processes. The next greatest potential impact from recycling is for NaNiCl₂ batteries. NaS and lithium battery recycling appears to be relatively low impact. The NiMH batteries that are recycled by technologies under development rather than pyrometallurgical processes also scored relatively well.

On an overall health/hazard basis, NiMH and advanced lithium batteries have much less impact on the general population than conventional lead-acid batteries. Lead-acid batteries will inevitably be in conventional cars as well as electric vehicles in the near term (1998-2005); for these batteries, the electrowinning process clearly presents the least anticipated impact for recycling the spent batteries.

NiMH batteries are a potential mid-term battery (2005-2010) for their increased energy density and extended range over the lead-acid batteries. Currently, the only process developed for recycling this battery is a pyrometallurgical process used at the INMETCO recycling facility in Pennsylvania. While this facility primarily recycles NiCd batteries, they do accept small amounts of NiMH consumer batteries as well. Cleaner processes for recovering metals in this battery are under development by A.D. Little, Teledyne Wah Chang Albany, and the U. S. Bureau of Mines. The process being developed by A.D. Little was used in this analysis for estimating emissions rates from the chemical processes. Compared to the pyrometallurgical process used by INMETCO, the chemical processes present a lesser impact in most categories. If these batteries do carry a significant percentage of the EV market in the mid-term, more research should be done to refine these chemical processes so that they may be developed on a commercial level.

Lithium-based batteries, such as the lithium-polymer and lithium-ion batteries, appear to be the most promising advanced battery technologies for the long-term (beyond 2010). Fortunately, these batteries contain only small amounts of toxic or carcinogenic substances, and are presently recycled (or anticipated to be recycled) by a clean process. The current versions of lithium-ion batteries contain relatively high amounts of nickel, but in advanced versions, the nickel will be almost eliminated and replaced with manganese, thus reducing the health impact of this battery. Estimated emissions rates from recycling these batteries may be low. The emissions to air shown in Section 2 are based on estimates from Toxco, the lithium battery recycler in British Columbia, Canada. No actual emissions data were available to verify those estimates, which often included zero emissions to air. Because Toxco uses a wet process, the results of this analysis were actually calculated for the final result assuming emissions rates typical of electrowinning processes, to be more conservative. If this battery is to be a serious long-term contender, this process, and others that may be under development, should be studied to further determine the best process from an environmental and economic standpoint.

4.1.2 Factors that Drive the Rankings

For smelted PbA batteries, the relatively high impact score, coupled with high g/mi of spent batteries to process and a moderate (0.002 percent) emissions rate combine to make a total health/hazard score dominated by lead. Although lead is also the driver when the spent batteries are processed by electrowinning, the very low emissions rate for the electrowinning process makes the total score much lower than when the same batteries are smelted.

The total score for NiCd batteries, which also contain cobalt and lithium, is driven by nickel and cadmium. Both have high raw scores and contain a moderate amount of material to process. Even the moderate emissions rate of 0.0006 percent cannot fully compensate for the high raw scores. Impacts from NiMH batteries are driven by nickel and iron. While iron is relatively benign, it is present in some of these batteries in a relatively high amount (about 3 g/mi), and for pyrometallurgical processing, may be emitted at a moderate rate.

The Na-NiCl₂ battery score is driven by the aluminum present in the electrolyte. Aluminum has a moderate impact, but a relatively high (0.05 percent) is emitted to the air during processing. Lithium-

polymer batteries have no clear driver, while lithium-ion batteries are driven by the nickel contained in the batteries. It is emitted at very low rates, but is present in the spent batteries at almost 2.3 g/mi.

Finally, zinc and nickel drive the zinc-air batteries. Zinc emissions rates are assumed to be moderate, based on secondary smelting operations for zinc in California. This, coupled with a moderate amount of zinc to process, results in a ranking in the midrange for these batteries if recycled through smelting processes.

4.2 Flammability/Reactivity Scores

Companies involved in battery transport and recycling need to ensure that flammability, corrosion, and reactivity are taken into account and minimized in transport, handling, and dismantling operations. This evaluation is only intended to cover the impacts posed by these batteries during the recycling and disposal process, and is not intended to cover vehicle operator accidents; however, such issues are taken into account in the design of the electric vehicle and in the development of emergency response procedures and training. Flammable and reactive solid wastes, such as lithium and sodium that are not recovered, will be treated and disposed of as non-flammable, nonreactive salts. Similarly, reactive wastes that enter the wastewater stream, such as sulfuric acid, must be neutralized before they can be discharged to public waters. While flammability and reactivity are addressed within the total health/hazard score, this section addresses them in more detail separately, as they may impact workers in the battery recycling field.

4.2.1 Flammability

Table 32 shows the weighted flammability for the spent battery pack, including the plastic casing. This was calculated by multiplying the flammability score of each constituent by its presence in the spent battery, and summing for each battery. The lithium batteries tend to have the highest flammability ratings (because of the flammability of lithium), while the nickel and zinc batteries have a low flammability potential. The moderate flammability of lead-acid batteries is due to their plastic casing. Other batteries also have flammable plastic cases. However, PbA batteries have a relatively short useful life, so more spent batteries are generated by vehicles powered by PbA batteries than by those utilizing other power sources. Therefore, more total plastic is generated per mile traveled. Table 33 shows how removing the impact of plastic from the analysis results in PbA batteries joining the “safest” list.

Table 32. Weighted Flammability Potential, Including Plastic

Battery Type	Flammability Potential	Battery Type	Flammability Potential
LiFeS	6.5	NaNiCl ₂	2.3
Li-Polymer	4.6	NiCd	2.1
LiFeS ₂	4.5	NaS	2.1
Li-ion	3.4	NiMH AB ₂	1.2
PbA	3.4	NiMH AB ₅	1.2
SBLA	2.7	Zn-Air	1.0

Table 33. Weighted Flammability Potential, Excluding Plastic

Battery Type	Flammability Potential
LiFeS	2.8
NaNiCl₂, LiFeS₂	2.3
NaS	2.1
Li-Polymer	2.0
Li-ion	0.4
All others	0.0

4.2.2 Reactivity

Table 34 shows the results of the reactivity ranking. The large amount of reactive electrolyte in spent flooded PbA batteries dominates the result. Sulfuric acid electrolyte is much more reactive than potassium hydroxide electrolyte. In addition, as previously mentioned, more spent batteries are generated using PbA technology than with other types. The nickel and zinc batteries are still ranked as relatively stable. Table 35 shows the extent of the electrolyte domination. Excluding electrolytes from the analysis results in lithium and sodium batteries having the highest reactivity potential.

Table 34. Weighted Reactivity Potential

Battery Type	Reactivity Potential	Battery Type	Reactivity Potential
PbA	10.7	LiFeS ₂	2.3
SBLA	6.3	NiCd	1.9
Li-Polymer	4.0	Li-ion	1.7
LiFeS	3.3	Zn-Air	1.0
NaS	3.0	NiMH AB ₂	0.7
NaNiCl ₂	2.5	NiMH AB ₅	0.7

Table 35. Weighted Reactivity Potential, Excluding Electrolyte

Battery Type	Reactivity Potential
Li-Polymer	2.0
NaNiCl ₂	1.8
LiFeS, Li-ion	1.3
LiFeS ₂	1.1
NaS	0.9
PbA	0.5
SBLA	0.1
All others	0.0

4.3 Uncertainty

It is important to note that there is a great deal of uncertainty surrounding this analysis. While the battery constituents are reasonably well known, although they vary from maker to maker and change somewhat as battery development progresses, recycling technologies for many of these batteries are in their infancy. While smelting technology for recycling lead-acid batteries is well developed, recycling by electrowinning processes is still under research development. For many other battery types, the assessment was performed using bench-scale or pilot plant technology. In some cases, actual emissions have been measured, while in other cases engineering judgement had to be utilized to adapt emission factors from other presumably similar processes. In addition, there can be substantial uncertainty surrounding the health impact values (cancer potency factors, MCLs, etc.). These numbers are based on controlled animal or human epidemiological studies that attempt to estimate human health impacts from environmental exposures. Among the uncertainties are extrapolating effects from animals to humans and drawing from experiments that use high doses of toxicants to predict impacts from low level environmental exposures. Because of the uncertainties built into the health/hazard scores, any interpretations made from this analysis should be viewed with caution.

4.4 Conclusions

Based on this health and hazard assessment effort, lead-acid batteries processed by smelting operations seem to have a larger potential negative health impact than the same batteries processed by other means or than other battery types. The NaNiCl_2 batteries also rank relatively more toxic. On an overall basis, the more advanced batteries such as NiMH, lithium-polymer, and lithium-ion represent a great improvement over conventional lead-acid battery technology, both in terms of battery performance and impacts from recycling the spent batteries. Results are graphically presented in Figure 2.

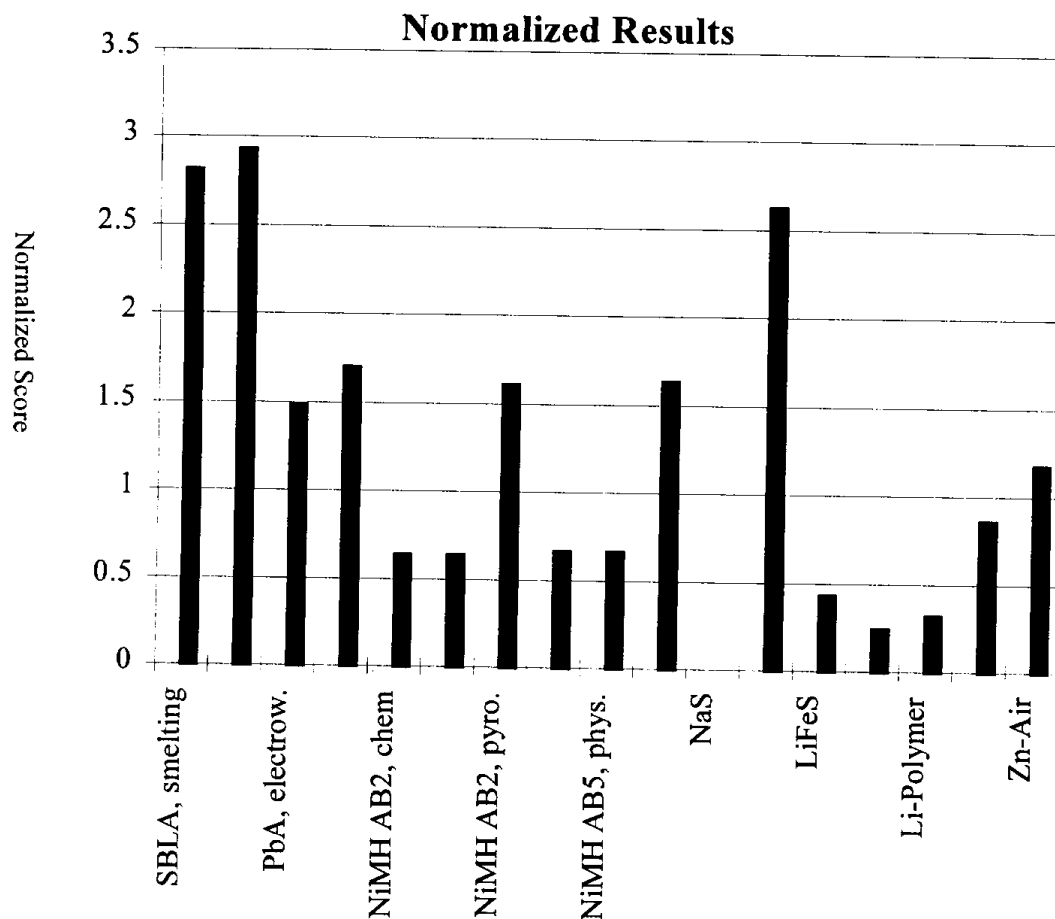


Figure 2. Normalized Results for Each Battery Type and Recycling Option

REFERENCES

1. Rand, D.A.J. "Batteries for Road Electric Vehicles: Systems in Competition with Nickel-Cadmium." *Nickel-Cadmium Battery Update '92*, the Cadmium Association, London, 1992.
2. Consultations with the Air Resources Board, Mobile Source Division, May 1994.
3. Cook, M. and H. Morrow. "Market Prospects for Nickel-Cadmium Batteries in Electric Vehicles." *Metal Bulletin Monthly*, April 1992.
4. Ovshinsky, S.R., M.A. Fetcenko, and J. Ross. "A Nickel Metal Hydride Battery for Electric Vehicles." *Science*, Vol. 20, April 9, 1993.
5. Consultation with Steve Albu, Mobile Source Division, California Air Resources Board, El Monte, California, September 29, 1994.
6. Consultations with William Meador, President, Recovery and Reclamation, Inc., Pecos, Texas, August, September, and October 1994.
7. Consultations with William McLaughlin, President, Toxco, Claremont, California, September and October 1994.
8. Sarre, G. , M. Broussely, and R. Staniewicz. "Lithium-Carbon (Lithium-Ion) Battery System for Electric Vehicles," presented at the 12th International Electric Vehicle Symposium, Anaheim, California, December 5-7, 1994.
9. Guyomard, D., and J.M. Tarascon. "Rocking-Chair or Lithium-Ion Rechargeable Lithium Batteries," *Advanced Materials*, Vol. 6, No. 5, 1994.
10. "Methodology for Estimating Emissions from On-Road Motor Vehicles, Volume II: EMFAC7F." California Air Resources Board, Technical Support Division of the Mobile Source Division, September 1993.
11. "Electric Vehicle Battery Material Safety Data Sheets." Arizona Public Service Company.
12. "Compilation of Air Pollutant Emission Factors, Stationary, Point, and Area Sources, AP-42," U.S. EPA, October 1986.
13. Mezey, E.J. "Characterization of Priority Pollutants from a Secondary Lead and Battery Manufacturing Facility," prepared by Battelle's Columbus Laboratories for U.S. EPA, EPA-600/2-79-039, January 1979.
14. "Nickel-Cadmium Batteries for Electric Vehicles: Life-Cycle Environmental and Safety Analysis," prepared by Geomet Technologies, Inc. for the Electric Power Research Institute, May 10, 1993.
15. "INMETCO: Making Metals a Reusable Resource." INMETCO Brochure, 1993.

16. Sabatini, J.C., E.L. Field, I-C. Wu, M.R. Cox, B.M. Barnett, and J.T. Coleman. "Feasibility Study for the Recycling of Nickel Metal Hydride Electric Vehicle Batteries." A.D. Little, Inc. for National Renewable Energy Laboratory, January 1994.
17. Consultations with James Rasmussen, Silent Power Ltd., at the Ad Hoc Electric Vehicle Battery Working Group, Recycling Sub-Working Meeting, Washington, D.C., August 29-30, 1994.
18. Consultations with Heinz Hammerling, Business Development Manager, AEG Corporation, Basking Ridge, New Jersey, September and October, 1994.
19. Abraham, K.M. and M. Alamgir. "Ambient temperature Rechargeable Polymer-Electrolyte Batteries." *Journal of Power Sources*, 43-44, pp.195-208, 1993.
20. "Waste Minimization Opportunity Assessment: A Phtofinishing Facility," prepared by Science Applications International Corporation for the U.S. EPA, EPA-600/2-91/039, August, 1991.
21. Wade, R., M. Williams, T. Mitchell, J. Wong, and B. Tuse. "Semiconductor Industry Study," California Department of Industrial Relations Division of Occupational Safety and Health, 1981.
22. "Methodology for Estimating Emissions from On-Road Motor Vehicles, Volume III: BURDEN7F." California Air Resources Board, Technical Support Division of the Mobile Source Division, September 1993.
23. Martin, C. "CalETC Utility EV Penetrations and Vehicle Splits." Letter from Cece Martin of the California Electric Transportation Coalition, February 26, 1993.
24. CAPCOA Air Toxics "Hot Spots" Program Revised 1992 Risk Assessment Guidelines, prepared by the Toxics Committee of the California Air Pollution Control Officers Association, October, 1993.
25. "Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children." EPA-540/R-93/081, February, 1994.
26. "GNB Incorporated AB2588 Toxic Hot Spots Health Risk Assessment," prepared by Environmental Audit, Inc., December, 1991.
27. "Proposed Identification of Inorganic Lead as a Toxic Air Contaminant, Part A: Exposure Assessment." California Air Resources Board, Stationary Source Division, August, 1993.
28. "Proposed Identification of Inorganic Arsenic as a Toxic Air Contaminant, Part A: Exposure Assessment." California Air Resources Board, Stationary Source Division, May, 1990.
29. California Environmental Protection Agency, Title 22, California Code of Regulations, Part 66261.24.
30. "National Fire Protection Association Hazardous Materials Ratings." *The Book of Chemical Lists*, Business and Legal Reports, Inc., 1993.
31. "Criteria for Carcinogens." Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, November 1, 1994.

32. "The Determination of Acute Toxicity Exposure Levels for Airborne Toxicants," a Draft for Public Comment, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, January, 1995.
33. Graedel, T.E., B.R. Allenby, P.R. Comrie. "Matrix Approach to Abridged Life Cycle Assessment. *Environmental Science and Technology*. " 29(3):134A-139A, 1995.
34. Allenby, B.R. "Integrating Environment and Technology; Design for Environment." in B.R. Allenby and D.J. Richards, eds. *The Greening of Industrial Ecosystems*. National Academy Press: Washington D.C., pp. 137-148, 1994.
35. Steele, N.L.C., D.T. Allen. "An Abridged Life-Cycle Assessment of Electric Vehicle Batteries." *Environ. Sci. Tech.*, **32**(1):40A-46A, 1998.
36. "Draft Staff Report: Proposed Update to the Toxic Air Contaminant List," Stationary Source Division, California Air Resources Board, December 1998.
37. Lewis, R.J., ed. *Hazardous Chemicals, Desk Reference*, 4th Ed., Van Nostrand Reinhold, 1997.
38. Cassaret and Doull's *Toxicology*, 3rd Ed., C. Klaassen, M. Amaur, and J. Doull, eds., 1986.
39. Ryan, R.P., C.E. Terry. *Toxicology Desk Reference*, 4th Ed., Taylor and Francis, Publisher, 1997.
40. "Technical Support Document for the Determination of Noncancer Chronic Reference Exposure Levels." Draft for Public Review. Cal/EPA Office of Environmental Health Hazard Assessment, October 1997.
41. Vimmerstedt, L.J., S. Ring, C.J. Hammel. "Current Status of Environmental Health and Safety Issues of Lithium-Ion Electric Vehicle Batteries." NREL Report No. TP-463-7673, September 1995.

LIST OF ACRONYMS

ARB	Air Resources Board. Division of the California Environmental Protection Agency which governs air quality regulations in California.
C_{ann}	Annual average ambient concentration ($\mu\text{g}/\text{m}^3$)
C_{max}	Maximum one-hour ambient concentration ($\mu\text{g}/\text{m}^3$)
CAPCOA	California Air Pollution Control Officers Association
CARB	California Air Resources Board. Division of the California Environmental Protection Agency which governs air quality regulations in California.
CFR	Cod of Federal Regulations
CO	Carbon Monoxide
Cr(III)	Trivalent chromium, non-toxic, non-carcinogenic
Cr(VI)	hexavalent chromium, toxic, carcinogenic
DOD	Depth of Discharge. This specifies how fully a battery is charged.
EPA	Environmental Protection Agency
EPRI	Electric Power Research Institute. A research organization funded by U.S. utility companies.
EV	Electric Vehicle
F_F	NFPA flammability rating
F_R	NFPA reactivity rating
FIRE	Factor Information Retrieval System
HCl	Hydrochloric acid leaching solution
HDV	Heavy Duty Vehicle
IARC	International Association for Research on Cancer
ICV	Internal Combustion Vehicle
IEUBK	Integrated Exposure Uptake Biokinetic Model
IRIS	Integrated Risk Information System
KOH	Potassium hydroxide electrolyte
LDV	Light Duty Vehicle
LiFeS	Lithium-iron sulfide battery
LiFeS ₂	Lithium-iron disulfide battery
Li-Ion	Lithium-ion battery
Li-Polymer	Lithium-polymer battery
M_{air}	Mass of pollutant emitted to air (g/mile)
M_{land}	Mass of pollutant disposed of on land (g/mile)
M_{water}	Mass of pollutant discharged to water (g/mile)
MCL	Maximum Contaminant Level
MCLG	Maximum Contaminant Level Goal
MDV	Medium Duty Vehicle
MRC	Metal Replacement Cartridges
MSDS	Material Safety Data Sheet
Na-NiCl ₂	Sodium-nickel chloride battery
NaS	Sodium-sulfur battery
NFPA	National Fire Protection Association
NiCd	Nickel-cadmium battery
NiMH	Nickel-metal hydride battery
NO _x	Oxides of nitrogen
NREL	National Renewable Energy Laboratory

OEHHA	Office of Environmental Health Hazard Assessment, A division of the California Environmental Protection Agency
Pb-Acid	Lead-acid battery
PE	Polyethylene
PP	Polypropylene
PS	Potency slope factor for cancer (mg/kg-day) ⁻¹
RAT	Relative acute toxicity risk
R _c	Relative cancer risk
RCT	Relative chronic toxicity risk
R _F	Relative flammability risk
R _L	Relative leachability
R _R	Relative reactivity risk
REL _i	Reference Exposure Level, inhalation basis (µg/m ³)
REL _o	Reference Exposure Level, oral basis (µg/m ³)
RHRAT	Relative Hazard Rating, acute toxicity
RHRC	Relative Hazard Rating, cancer
RHRCT	Relative Hazard Rating, chronic toxicity
RHR _F	Relative Hazard Rating, flammability
RHR _L	Relative Hazard Rating, leachability
RHR _R	Relative Hazard Rating, reactivity
S _F	Qualitative flammability score for each battery
S _R	Qualitative reactivity score for each battery
SO _x	Oxides of sulfur
STLC	Soluble Threshold Limit Concentration
TAC	Toxic Air Contaminant
TCLP	Toxicity Characteristic Leaching Procedure
TTLC	Total Threshold Limit Concentration
UR	Unit Risk Factor for cancer (µg/m ³) ⁻¹
USABC	United States Advanced Battery Consortium
UT _i	Inhalation uptake
UT _o	Oral uptake
UT _T	Total uptake
VMT	Vehicle Miles Traveled
ZEV	Zero Emission Vehicle
Zn-air	Zinc-air battery

APPENDIX A

Appendix A

BATTERY RECYCLING HEALTH & HAZARD IMPACT ASSESSMENT

(Maximum Score Achievable = 80 points)

This assessment format was developed specifically to enable the relative ratings of the impacts of recycling a variety of battery types that could be utilized in electric vehicles. The actual point values obtained should not be used for any purpose other than a ranking of this nature.

1. Carcinogenic Potential* (Max = 10 pts)

IARC Group 1 or EPA Group A	10
IARC Group 2A or EPA Group B1	8
IARC Group 2B or EPA Group B2	6
EPA Group C	4
IARC Group 3 or EPA Group D	2
IARC Group 4 or No known or anticipated cancer effects	0

2. Cancer Potency (m^3/ug) (inhalation) (Max = 10 pts)

$\geq 10^{-2}$	10
$\geq 10^{-3}$ but $< 10^{-2}$	8
$\geq 10^{-4}$ but $< 10^{-3}$	6
$\geq 10^{-5}$ but $< 10^{-4}$	4
$< 10^{-5}$	2
likely carcinogen but no unit risk determined	2
no indication of carcinogenicity	0

3. Cancer Potential (ingestion) (Max = 10 pts)

yes	10
maybe	5
no	0

4. Noncancer Impacts (inhalation) (RELs, ug/m^3) (Max = 10 pts)

≤ 0.5 or no identified threshold, but would likely be below 0.5	10
> 0.5 but ≤ 1.0	8
> 1.0 but ≤ 10	6
> 10 but ≤ 50	4
> 50 but ≤ 100	2
possible nonquantified impacts	2
> 100 , or no REL because no likely impacts of significance	0

* Appendix A1 includes a definition of these categories

5. Noncancer Impacts (ingestion) (MCL, or MCLG; ug/l) (Max = 10 pts)

< 10 ⁻³	10
10 ⁻³ to 10 ⁻²	8
10 ⁻² to 10 ⁻¹	4
nonquantified	2
>10 ⁻¹ or none likely or US EPA declared none was needed	0

6. Number of Known Organ Systems Affected (noncancer) (Max = 5 pts)

4 or more	5
3	4
2	3
1	2
unknown	1
No effect on an organ system considered significant	0

7. Chronic/Acute/Reproductive or Developmental Toxicity (noncancer) (Max = 5 pts)

Substance has all 3 of the listed effects	5
Substance has 2 of the 3 listed effects	4
Substance has 1 of the 3 listed effects	2
Unknown effects	1
No noncancer effects considered significant	0

8. Occupational Exposure Limits (PELs, ug/m3) (Max = 5 pts)

< 10 ⁻²	5
≥10 ⁻² but less than 0.1	4
≥10 ⁻¹ but less than 1.0	2
≥1.0 or not established	1
no established exposure limits because OSHA declared none was needed	0

9. Ecotoxicological Potential (Max = 10 pts, sum of A and B)

A. Chronic toxicity to aquatic organisms (USEPA AWQC or Suter Tier II, ug/l)

<10	5
≥10 but less than 10 ²	4
≥10 ² but less than 10 ³	3
No criteria; potential impacts, but insufficient data	2
≥ 10 ³	1
No criteria; potential impacts unlikely	0

B. Chronic toxicity to avian and mammalian wildlife (lowest NOAEL, mg/kg/d)	
<10	5
≥ 10 but less than 10^2	4
$\geq 10^2$ but less than 10^3	3
No criteria; potential impacts, but insufficient data	2
$\geq 10^3$	1
No criteria; potential impacts unlikely	0

10. Flammability/Reactivity (National Fire Prevention Association Rankings)

NFPAFlammability + NFPAReactivity > 4	5
NFPAFlammability + NFPAReactivity = 4	4
NFPAFlammability + NFPAReactivity = 3	3
NFPAFlammability + NFPAReactivity = 2	2
NFPAFlammability + NFPAReactivity = 1	1
NFPAFlammability + NFPAReactivity = 0	0

The Assessment Protocol

Data were gathered from a variety of sources to enable completion of the assessment protocol. The data and their sources are shown in Section 3, Table 28.

A raw impact score was generated for each battery constituent (e.g., cadmium), by applying the relevant data (carcinogenicity, toxicity, flammability, etc. from Table 28) to the ranking protocol. For instance, cadmium has an IARC classification of 1 for carcinogenic potential (Appendix A1). This corresponds to 10 points for Item 1. Another 8 points was generated based on its cancer potency of 4.2×10^{-3} (Item 2). It has no potential to be an oral carcinogen (0 points). The established REL of 3.5 generates another 6 points, and so on, until a total of 57 points (see Table 30) out of the possible 80 was generated.

This score was multiplied by the amount of cadmium present in the spent batteries (g/mile traveled) and by the emission rate of cadmium when the battery was recycled using the specified technology. For instance, cadmium is present in Sealed Bipolar Lead Acid (SBLA) batteries. There is about 0.04 g/mile generated that must be recycled when the battery is spent (See Table 4; The spent material to process is based on light-duty vehicle applications. However, the battery results in approximately 0.00091 percent of this cadmium being released to the air (See Tables 6-22). The contribution of cadmium to the ranking of this battery using the smelting process is:

$$57 * 0.04 * 0.00091\% = 2.07 \times 10^{-5}$$

This contribution is added to that of lead, copper, antimony, tin, sulfuric acid, and the plastic casing material used in this battery to generate a total impact of 1.24×10^{-2} . (There are no units to be concerned with, as the total scores were normalized. Nominally, the units would be g/mile spent battery material emitted to air.)

Data and results of the calculations are given in Appendix A2.

Appendix A1
IARC and EPA Cancer Group Classifications

IARC		EPA	
Group 1	Human carcinogen	Group A	Human carcinogen (sufficient epidemiological evidence)
Group 2A	Probable human carcinogen	Group B1	Probable human carcinogen (sufficient animal and limited human evidence)
Group 2B	Possible human carcinogen	Group B2	Probable human carcinogen (sufficient animal and no human evidence)
		Group C	Possible human carcinogen (limited animal and no human evidence)
Group 3	Not classifiable	Group D	Non classifiable (insufficient evidence, or no data available, in animals and humans)
Group 4	Probably not carcinogenic to humans	Group E	Evidence of non-carcinogenicity in humans

Appendix A2
Calculation of Battery Rankings

Battery	Substance	Mass (g/ml)	% to air	Carcin.	Potency	Oral Ccr	REL	MCL	Organ	Effect	PEL	AqTox	TerTox	FURtoxic	sum	Emissions	* score	log norm.
SBLA smelting	Pb	10.91	0.002000%	6	4	10	10	2	5	5	5	4	5	5	56	2.18E-04	1.22E-02	
	Cu	0.11	0.002730%	2	1	8	6	0	5	5	5	2	5	5	31	3.00E-06	9.31E-05	
	Cd	0.04	0.000910%	10	8	0	0	8	5	5	5	5	5	0	57	3.64E-07	2.07E-05	
	Sb	0.05	0.001000%	6	2	5	10	8	3	4	4	2	4	5	51	5.00E-07	2.55E-05	
	Sn	0.03	0.000700%	0	0	0	0	0	3	2	2	2	4	2	13	2.10E-07	2.73E-06	
Plastic case		0.81	0.000000%	0	0	0	0	0	4	4	4	1	0	0	3	0.00E+00	0.00E+00	
	H2SO4	3.11	0.000100%	0	0	0	0	0	4	4	4	1	0	2	11	3.11E-06	3.42E-05	
Pb-Acid smelting	Total																1.24E-02	2.81
	Pb	13.64	0.002000%	6	4	10	10	2	5	5	5	4	5	5	56	2.73E-04	1.53E-02	
	Sb	0.45	0.001000%	6	2	5	10	8	4	4	4	2	4	5	51	4.50E-06	2.30E-04	
	As	0.05	0.000730%	10	8	10	10	4	4	4	5	4	4	5	65	3.65E-07	2.37E-05	
	Sn	0.05	0.000700%	0	0	0	0	0	3	2	2	2	4	2	13	3.50E-07	4.55E-06	
Plastic case		1.14	0.018000%	0	0	0	0	0	4	4	4	1	0	0	3	2.05E-04	6.16E-04	
	H2SO4	5.09	0.000100%	0	0	0	0	0	4	4	4	1	0	2	11	5.09E-06	5.60E-05	
Pb-Acid electrow	Total																1.62E-02	2.93
	Pb	13.64	0.000666%	6	4	10	10	2	5	5	5	4	5	5	56	9.00E-06	5.04E-04	
	Sb	0.45	0.000666%	6	2	5	10	8	4	4	4	2	4	5	51	2.97E-07	1.51E-05	
	As	0.05	0.000666%	10	8	10	10	4	4	4	5	4	4	5	65	3.30E-08	2.15E-06	
	Sn	0.05	0.000666%	0	0	0	0	0	3	2	2	2	4	2	13	3.30E-08	4.29E-07	
Plastic case		1.14	0.000000%	0	0	0	0	0	4	4	4	1	0	0	3	0.00E+00	0.00E+00	
	H2SO4	5.09	0.000100%	0	0	0	0	0	4	4	4	1	0	2	11	5.09E-06	5.60E-05	
NiCd Pyromet	Total																5.78E-04	1.48
	Ni	1.66	0.000600%	10	6	0	10	0	4	4	5	1	5	4	45	9.96E-06	4.48E-04	
	Cd	1.17	0.000666%	10	8	0	6	8	5	5	5	5	5	5	57	7.79E-06	4.44E-04	
	Co	0.01	0.000600%	6	2	0	4	2	5	5	4	2	5	5	35	6.00E-08	2.10E-06	
	Li	0	0.000600%	0	0	0	4	4	5	4	4	2	2	2	25	0.00E+00	0.00E+00	
Electrolyte (KOH)	Casing	0.71	0.000600%	0	0	0	0	0	1	2	2	1	0	0	0	4.26E-06	0.00E+00	
	Electrolyte (KOH)	1.88	0.000600%	0	0	0	0	0	1	2	2	1	0	1	5	1.13E-05	5.64E-05	
	Total																9.51E-04	1.70
	Ni	1.82	0.000666%	10	6	0	10	0	4	4	5	1	5	4	45	1.20E-06	5.41E-05	
	Fe	3.3	0.000666%	0	0	0	0	0	4	4	2	1	1	1	8	2.18E-06	1.74E-05	
NIMH, AB2 chemical	V	0.54	0.000666%	0	0	0	4	4	4	4	2	2	2	0	18	3.56E-07	6.42E-06	
	Zr	0.19	0.000666%	0	0	0	0	0	1	1	1	1	4	0	7	1.25E-07	8.78E-07	
	Ti	0.06	0.000666%	0	0	0	0	0	2	1	1	1	4	0	4	3.96E-08	1.58E-07	
	Cr (III)	0.16	0.000666%	2	1	5	0	4	4	4	2	3	5	0	30	1.06E-07	3.17E-06	
	Al	0.03	0.000666%	0	0	0	0	0	4	4	4	1	4	5	20	1.98E-08	3.96E-07	
Polypropylene	KOH	0.68	0.000000%	0	0	0	0	0	1	2	2	1	4	1	5	0.00E+00	0.00E+00	
	Polypropylene	0.38	0.000000%	0	0	0	0	0	1	2	2	1	4	1	3	0.00E+00	0.00E+00	
	Total																8.25E-05	0.63
	Ni	1.82	0.000666%	10	6	0	10	0	4	4	5	1	5	4	45	1.20E-06	5.41E-05	
	Fe	3.3	0.000666%	0	0	0	0	0	4	4	2	1	1	1	8	2.18E-06	1.74E-05	
NIMH, AB2 phys/chem	V	0.54	0.000666%	0	0	0	4	4	4	4	2	2	2	0	18	3.56E-07	6.42E-06	
	Zr	0.19	0.000666%	0	0	0	0	0	1	1	1	1	4	0	7	1.25E-07	8.78E-07	
	Ti	0.06	0.000666%	0	0	0	0	0	2	1	1	1	4	0	4	3.96E-08	1.58E-07	
	Cr (III)	0.16	0.000666%	2	1	5	0	4	4	4	2	3	5	0	30	1.06E-07	3.17E-06	
	Al	0.03	0.000666%	0	0	0	0	0	4	4	4	1	4	5	20	1.98E-08	3.96E-07	
Polypropylene	KOH	0.68	0.000000%	0	0	0	0	0	1	2	2	1	4	1	5	0.00E+00	0.00E+00	
	Polypropylene	0.38	0.000000%	0	0	0	0	0	1	2	2	1	4	1	3	0.00E+00	0.00E+00	
Total																	8.25E-05	0.63

Appendix A2
Calculation of Battery Rankings

Battery	Substance	Mass (g/ml)	% to air	Carcin.	Potency Oral Ccr	REL	MCL	Organ	Effect	PEL	AqTox	TerrTox	FIRReac.	sum	Emissions	* score	log norm.
NIMH, AB2 Pyromet.	Ni	1.82	0.000600%	10	6	0	10	0	4	5	1	5	4	0	45	1.09E-05	4.91E-04
	Fe	3.3	0.000600%	0	0	0	0	0	4	2	1	1	0	0	8	1.98E-05	1.58E-04
	V	0.54	0.000600%	0	0	0	4	0	4	4	2	2	2	0	18	3.24E-06	5.89E-05
	Zr	0.19	0.000600%	0	0	0	0	0	1	1	1	4	0	0	7	1.14E-06	7.98E-06
	Ti	0.06	0.000600%	0	0	0	0	2	2	1	1	1	0	0	4	3.60E-07	1.44E-06
	Cr (III)	0.16	0.000600%	2	1	5	0	4	4	4	2	3	5	0	30	9.60E-07	2.88E-05
	Al	0.03	0.000600%	0	0	0	0	0	4	4	1	4	5	2	20	1.80E-07	3.60E-06
	KOH	0.68	0.000600%	0	0	0	0	0	1	2	1	0	0	1	5	4.08E-06	2.04E-05
	Polypropylene	0.38	0.000000%	0	0	0	0	0	0	0	0	0	0	3	3	0.00E+00	0.00E+00
	Total															7.70E-04	1.60
NIMH, AB5 Chemical	Ni	2.21	0.000666%	10	6	0	10	0	4	5	1	5	4	0	45	1.46E-06	6.56E-05
	Fe	3.3	0.000666%	0	0	0	0	0	4	2	1	1	0	0	8	2.18E-06	1.74E-05
	Co	0.13	0.000666%	6	2	0	4	2	5	4	2	5	5	0	35	8.58E-08	3.00E-06
	Mn	0.06	0.000666%	2	1	0	10	2	5	4	1	4	4	0	33	3.96E-08	1.31E-06
	Al	0.02	0.000666%	0	0	0	0	0	4	4	1	4	5	2	20	1.32E-08	2.64E-07
	KOH	0.68	0.000000%	0	0	0	0	0	1	2	1	0	0	1	5	0.00E+00	0.00E+00
	Polypropylene	0.38	0.000000%	0	0	0	0	0	0	0	0	0	0	3	3	0.00E+00	0.00E+00
	Total															8.76E-05	0.66
	Ni	2.21	0.000666%	10	6	0	10	0	4	5	1	5	4	0	45	1.46E-06	6.56E-05
	Fe	3.3	0.000666%	0	0	0	0	0	4	2	1	1	0	0	8	2.18E-06	1.74E-05
NIMH, AB5 Pyromet.	Co	0.13	0.000666%	6	2	0	4	2	5	4	2	5	5	0	35	8.58E-08	3.00E-06
	Mn	0.06	0.000666%	2	1	0	10	2	5	4	1	4	4	0	33	3.96E-08	1.31E-06
	Al	0.02	0.000666%	0	0	0	0	0	4	4	1	4	5	2	20	1.32E-08	2.64E-07
	KOH	0.68	0.000000%	0	0	0	0	0	1	2	1	0	0	1	5	0.00E+00	0.00E+00
	Polypropylene	0.38	0.000000%	0	0	0	0	0	0	0	0	0	0	3	3	0.00E+00	0.00E+00
	Total															8.76E-05	0.66
	Ni	2.21	0.000600%	10	6	0	10	0	4	5	1	5	4	0	45	1.33E-05	5.97E-04
	Fe	3.3	0.000600%	0	0	0	0	0	4	2	1	1	0	0	8	1.98E-05	1.58E-04
	Co	0.13	0.000600%	6	2	0	4	2	5	4	2	5	5	0	35	7.80E-07	2.73E-05
	Mn	0.06	0.000600%	2	1	0	10	2	5	4	1	4	4	0	33	3.60E-07	1.19E-05
Na-S Silent Powe Process	Al	0.02	0.000600%	0	0	0	0	0	4	4	1	4	5	2	20	1.20E-07	2.40E-06
	KOH	0.68	0.000600%	0	0	0	0	0	1	2	1	0	0	1	5	4.08E-06	2.04E-05
	Polypropylene	0.38	0.000000%	0	0	0	0	0	0	0	0	0	0	3	3	0.00E+00	0.00E+00
	Total															8.17E-04	1.63
	Na	0.44	0.000100%	0	0	0	6	3	3	4	1	2	2	5	23	4.40E-07	1.01E-05
	S, as H2SO4	0.82	0.000100%	0	0	0	0	4	4	4	1	0	0	2	11	8.20E-07	9.02E-06
	Alpha alumina ele	1.06	0.000000%	0	0	0	0	0	0	0	0	0	0	0	0	0.00E+00	0.00E+00
	Beta alumina elect	1.06	0.000000%	0	0	0	0	0	0	0	0	0	0	0	0	0.00E+00	0.00E+00
	Cell case	0.53	0.000000%	0	0	0	0	0	0	0	0	0	0	0	0	0.00E+00	0.00E+00
	Carbon fiber/graph	0.07	0.000000%	6	4	0	0	4	4	4	2	0	0	0	20	0.00E+00	0.00E+00
Na-NiCl2 AEG metho	Total															1.91E-05	0.00
	Na	0.52	0.000100%	0	0	0	6	3	3	4	1	2	2	5	23	5.20E-07	1.20E-05
	Ni	0.66	0.000600%	10	6	0	10	0	4	5	1	5	4	0	45	3.96E-06	1.78E-04
	Cl	0.49	0.000100%	0	0	0	6	2	3	4	1	5	2	0	23	4.90E-07	1.13E-05
	Al	0.78	0.050000%	0	0	0	0	0	4	4	1	4	5	2	20	3.90E-04	7.80E-03
	Steel casing	1.89	0.000000%	0	0	0	0	0	0	0	0	0	0	0	0	0.00E+00	0.00E+00
	Total															8.00E-03	2.62
	Na	0.52	0.000100%	0	0	0	6	3	3	4	1	2	2	5	23	5.20E-07	1.20E-05
	Ni	0.66	0.000600%	10	6	0	10	0	4	5	1	5	4	0	45	3.96E-06	1.78E-04
	Cl	0.49	0.000100%	0	0	0	6	2	3	4	1	5	2	0	23	4.90E-07	1.13E-05

Appendix A2
Calculation of Battery Rankings

Battery	Substance	Mass (g/ml)	% to air	Carcin.	Potency	Oral Ccr	REL	MCL	Organ	Effect	PEL	AqTox	TerrTox	F/Reac.	sum	Emissions	* score	log norm.
Li-FeS Toxco meth	Li	0.66	0.000100%	0	0	0	4	4	5	4	4	2	2	4	25	6.80E-07	1.85E-05	
	Fe	2.65	0.000100%	0	0	0	0	0	4	2	2	1	1	0	0	2.65E-06	2.12E-05	
	S	1.51	0.000100%	0	0	0	0	0	4	2	1	1	1	0	8	2.65E-06	1.36E-05	
	Electrolyte	2.01	0.000100%	0	0	0	0	0	1	1	2	2	2	1	9	1.51E-06	0.00E+00	
	Casing (Teflon)	1.21	0.000000%	0	0	0	0	0	0	0	0	0	0	0	0	2.01E-06	0.00E+00	
Li-FeS2 Toxco meth	Total															0.00E+00	0.00E+00	
	Li	0.54	0.000100%	0	0	0	4	4	5	4	4	2	2	4	25	5.40E-07	5.13E-05	0.43
	Fe	1.07	0.000100%	0	0	0	0	0	4	2	2	1	1	0	8	1.07E-06	1.35E-05	
	S	1.25	0.000100%	0	0	0	0	0	1	1	2	2	2	1	9	1.25E-06	8.96E-06	
	Electrolyte	1.19	0.000100%	0	0	0	0	0	0	0	0	0	0	0	0	1.19E-06	1.13E-05	
Li-Polymer Toxco meth	Casing (Teflon)	0.72	0.000000%	0	0	0	0	0	0	0	0	0	0	0	0	0.00E+00	0.00E+00	
	Li	0.99	0.000100%	0	0	0	4	4	5	4	4	2	2	4	25	9.90E-07	3.33E-05	0.24
	Polymer (electrolyt)	1.98	0.000100%	0	0	0	0	0	0	0	0	0	0	0	0	1.98E-06	2.48E-05	
	Polyethylene case	0.86	0.000100%	0	0	0	0	0	0	0	0	0	0	0	0	8.60E-07	0.00E+00	
	Cu	0.4	0.000100%	2	1	1	6	6	5	5	5	2	5	0	31	4.00E-07	1.24E-05	
Li-Ion Toxco meth	Total																3.97E-05	
	Li	0.14	0.000100%	0	0	0	4	4	5	4	4	2	2	4	25	1.40E-07	3.50E-06	
	Ni	2.29	0.000100%	10	6	0	10	10	4	4	5	1	5	4	45	2.29E-06	1.03E-04	
	P	0.06	0.000100%	2	1	1	10	10	5	4	2	2	2	5	33	6.00E-08	1.98E-06	
	F	0.22	0.000100%	2	1	1	0	0	2	1	4	2	1	4	22	2.20E-07	4.84E-06	
C. graphite Organic electrolyte	C. graphite	1.17	0.000100%	6	4	0	0	0	4	4	4	2	0	0	20	1.17E-06	2.34E-05	
	Organic electrolyte	0.39	0.000100%	0	0	0	0	0	0	0	0	0	0	0	0	3.90E-07	0.00E+00	
	Teflon	0.96	0.000000%	0	0	0	0	0	0	0	0	0	0	0	0	0.00E+00	0.00E+00	
	Total																1.37E-04	
	Zn	1.39	0.000640%	2	1	1	4	4	3	2	2	1	4	4	21	8.90E-06	1.87E-04	0.85
Zinc-Air R&R	Ni	0.35	0.000600%	10	6	0	10	10	4	5	1	5	4	0	45	2.10E-06	9.45E-05	
	KOH	1.04	0.000000%	0	0	0	0	0	1	2	2	1	1	1	5	0.00E+00	0.00E+00	
	Polypropylene, PE	0.35	0.000000%	0	0	0	0	0	0	0	0	0	0	0	0	0.00E+00	0.00E+00	
	Teflon	0.1	0.000000%	0	0	0	0	0	0	0	0	0	0	0	0	0.00E+00	0.00E+00	
	Lamp black carbo	0.07	0.000000%	6	4	0	0	0	4	4	4	2	0	0	20	0.00E+00	0.00E+00	
Source:	Total																2.81E-04	
																		1.17
The health scores are from Table 27 and Appendix A															Sum of Col 5-15	% to air times g/ml	Emissions times sum	
Table 4																		
Tables 5-22																		

APPENDIX B

CARCINOGENIC AND NON-CARCINOGENIC TOXICOLOGY EVALUATION



ENVIRONMENTAL RESEARCH, INC.

1950 ADDISON STREET • SUITE 202 • BERKELEY, CA 94704 • TELEPHONE (510) 845-8484

Carcinogenic and Non-Carcinogenic Toxicology
of
11 Chemicals Associated with Automobile Batteries

6 February 1995

Prepared by
Joseph M. Rut
&
Selina Bendix, Ph.D., R.E.A

TABLE OF CONTENTS

Information Sources	1
Methods	3
Chemical Information	
Table 1: Summary of Carcinogenic and Non-Carcinogenic Toxicology	8
Table 2: Cancer Risk and Lethal Dose/Concentration	9
Cadmium	10
Dioxin	17
Iron	22
Lead	25
Lithium	31
Nickel	33
Sodium	39
Sulfur	41
Sulfuric Acid	43
Vanadium	46
Zinc	48
Endnotes	51

INFORMATION SOURCES

The main references used to compile the information in this report are listed below, preceded by the abbreviations used to identify them, in the text of this report, as the specific sources of information. Information on immunotoxicity was gathered largely from Bendix Environmental Research, Inc.'s proprietary database. References for immunotoxicity and some other information appear as endnotes to this report.

1991 ACGIH: American Conference of Governmental Hygienists, Inc. (1991) *Documentation of the Threshold Limit Values and Biological Exposure Indices, 6th Edition*, American Conference of Governmental Hygienists, Inc., Cincinnati, OH.

1993-1994 ACGIH: American Conference of Governmental Hygienists, Inc. (1993) *1993-1994 Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices*, American Conference of Governmental Hygienists, Inc., Cincinnati, OH.

CAPCOA: California Air Pollution Control Officers Association (1993) *Air Toxics "Hot Spots" Program, Revised 1992 Risk Assessment Guidelines*.

EPA: United States Environmental Protection Agency (1987) *Health Assessment Document for Beryllium, EPA/600/8-84/026F*, Office of Health and Environmental Assessment, Washington, D.C. (This document, the most recent in the series, contains a summary table of previous carcinogenicity assessments for other chemicals.)

EPA2: United States Environmental Protection Agency (1994) *Health Assessment Document for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds*, Office of Health and Environmental Assessment, Washington, D.C.

Gosselin: Gosselin, Robert E., et al. (1984) *Clinical Toxicology of Commercial Products, 5th Edition*, Williams & Wilkins, Baltimore/London.

HEAST: United States Environmental Protection Agency (1994) *Health Effects Assessment Summary Tables (HEAST), FY-1994 Annual*, Office of Solid Waste and Emergency Response, Washington, D.C.

IARC Supplement 7: International Agency for Research on Cancer (1987) *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Volumes 1 to 42, Supplement 7*, International Agency for Research on Cancer, Lyon, France.

IRIS: United States Environmental Protection Agency (1994) *Integrated Risk Information System (IRIS)*, On-line search of computer database performed December 1994.

Jim Collins, OEHHHA: Phone conversation between Jim Collins, California EPA, Office of Environmental Health Hazard Assessment (OEHHHA), and Joseph Rut, Bendix Environmental Research, Inc., 12 December 1994.

Lewis: Lewis, Richard J., Sr. (1991) *Reproductively Active Chemicals*, Van Nostrand Reinhold, New York.

Meeks: Meeks, Robert G., et al. (1991) *Hepatotoxicology*, CRC Press, Boca Raton, Florida.

NIOSH: United States Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health (1990) *NIOSH Pocket Guide to Chemical Hazards*.

OEHHHA: California Environmental Protection Agency, Office of Environmental Health Hazard Assessment (1994) *California Environmental Protection Agency Criteria for Carcinogens*.

RTECS: United States Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health (1994) *Registry of Toxic Effects of Chemical Substances*, On-line search of computer database performed December 1994.

Sax: Lewis, Richard J., Sr., Ed. (1992) *Sax's Dangerous Properties of Industrial Materials*, 8th Edition, Van Nostrand Reinhold, New York.

Zimmerman: Zimmerman, Hyman J. (1978) *Hepatotoxicity: The Adverse Effects of Drugs and Other Chemicals on the Liver*, Appleton-Century-Crofts, New York.

METHODS

This report is an assessment of the carcinogenic and non-carcinogenic toxicology of 11 chemicals potentially associated with automotive batteries and automotive battery recycling technology. The 11 chemicals were chosen by Acurex Environmental Corporation and assigned to Bendix Environmental Research, Inc. for assessment. This assessment is based on the elemental or primary form of the 11 chemicals and does not include all compound forms of the chemicals. Information on some compound forms of the chemicals has been included where it was readily available in the sources searched for information on the elemental or primary forms.

For each chemical, specific information regarding carcinogenicity and non-carcinogenic toxicology has been gathered. The methodology and/or sources for each category of information follow, in the order in which information appears for each chemical in the text of this document.

Carcinogenicity

Overall Carcinogenicity Rating: For the purpose of this assessment the IARC rating, arrived at by consensus of an international work group, is given precedence over the EPA rating if both entities have rated the chemical. If neither IARC nor EPA have rated the chemical, a rating of 3 (not classifiable) is assigned. To keep ratings numerical, IARC 2B can be represented as IARC 2.5. In general, IARC ratings correspond to EPA ratings as follows:

Human carcinogen	IARC 1 = EPA A
Probable human carcinogen	IARC 2A = EPA B1
Possible/probable human carcinogen	IARC 2B = EPA B2 or EPA C, depending upon strength of evidence.
Not classifiable (inadequate data)	IARC 3 = EPA D
Probably not carcinogenic	IARC 4 = EPA E

There is some overlap between IARC rating 2B and EPA ratings B2 and C.

The EPA B2 rating is usually based on sufficient evidence of carcinogenicity in animals with lack of or inadequate data in humans. EPA designates this level of data as evidence of "probable" carcinogenicity in humans.

The EPA C rating is based on limited evidence of carcinogenicity in animals with lack of or inadequate data in humans. EPA designates this level of data as evidence of "possible" carcinogenicity in humans.

The IARC 2B rating can include chemicals with either of the levels of data which would assign them an EPA B2 or C rating, as well as chemicals with limited evidence of carcino-

genicity in humans with lack of or inadequate evidence of carcinogenicity in animals. IARC designates all these levels of data as evidence of "possible" carcinogenicity in humans.

IARC Cancer Rating: Source: IARC Supplement 7, a summary of the *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*.

IARC Cancer Ratings are as follows:

- Group 1 - Human carcinogen (Sufficient evidence of carcinogenicity in humans.)
- Group 2A - Probable human carcinogen (Limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in experimental animals. On occasion, IARC may classify an agent in this category solely on the basis of limited evidence of carcinogenicity in humans or of sufficient evidence of carcinogenicity in experimental animals in view of supporting evidence from other relevant data.)
- Group 2B - Possible human carcinogen (This category usually includes agents for which there is limited evidence in humans in the absence of sufficient evidence in experimental animals. It may also be used when there is inadequate evidence of carcinogenicity in humans or when human data is non-existent but there is sufficient evidence of carcinogenicity in experimental animals. In some instances, agents may be included for which there is inadequate evidence or no data in humans but limited evidence of carcinogenicity in experimental animals together with supporting evidence from other relevant data.)
- Group 3 - Not classifiable as to human carcinogenicity (Agents are placed in this group when they do not fall into any other group.)
- Group 4 - Probably not a human carcinogen (Evidence suggests lack of carcinogenicity in humans together with evidence suggesting lack of carcinogenicity in experimental animals. In some circumstances, agents for which there is inadequate evidence of or no data in carcinogenicity in humans but evidence suggesting lack of carcinogenicity in experimental animals, consistently and strongly supported by a broad range of other relevant data, may be classified into this group.)

EPA Cancer Weight-of-Evidence Group: The primary source for this information was the EPA's IRIS database, which is the most up-to-date posting of EPA assessments of chemicals. If no posting was found on IRIS, secondary checks were made to EPA's HEAST document and a 1987 EPA list of chemicals assessed for carcinogenicity.^a The basis for the EPA rating was included when available.

EPA Weight-of-Evidence Groups are as follows:

^a Received from Jim Coglianò, Chief, Carcinogen Assessment Statistics and Epidemiology Branch, Office of Health and Environmental Assessment (OHEA). Mr. Coglianò states that the list will soon be updated to include more chemicals and that the IRIS database is the most up-to-date source for EPA ratings.

- Group A - Human carcinogen (sufficient evidence of carcinogenicity in humans).
- Group B - Probable human carcinogen (B1 - limited evidence of carcinogenicity in humans; B2 - sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans).
- Group C - Possible human carcinogen (limited evidence of carcinogenicity in animals and inadequate or lack of data in humans).
- Group D - Not classifiable as to human carcinogenicity (inadequate or no evidence).
- Group E - Evidence of noncarcinogenicity for humans (no evidence of carcinogenicity in adequate studies).

Inhalation Cancer Potency Value: Sources: IRIS, CAPCOA, OEHHA and HEAST. Potency values obtained from HEAST are still preliminary. This value is either a Potency Slope^b or Unit Risk^c for a material administered via the inhalation route.

Non-Inhalation Cancer Potency Value: Sources: IRIS, CAPCOA, OEHHA and HEAST. Potency values obtained from HEAST are still preliminary. This value is either a Potency Slope or Unit Risk for a material administered via the non-inhalation route.

Carcinogenic Potential: Sources: RTECS, IRIS and Sax. Evidence compiled for carcinogenic potential includes studies showing carcinogenic, neoplastic, or tumorigenic effects. Equivocal evidence was only entered if unequivocal evidence was not available.

Mutagenic Potential: Sources: RTECS, IRIS and Sax. RTECS and Sax only list studies positive for mutagenicity. IRIS lists tests both positive and negative for mutagenicity.

NON-CARCINOGENIC TOXICOLOGY

Overall Non-carcinogenic

Toxicology Rating: The overall non-carcinogenic toxicology of the chemicals is rated on a scale of 1 to 5. Like the overall carcinogenicity rating, lower numbers represent a greater hazard. Chemicals posing an acute hazard (e.g. metallic sodium, metallic lithium, and sulfuric acid all pose direct burn hazards on contact with skin) are rated a 1, as are chemicals with chronic effects which occur at very low levels. Chemicals with chronic effects occurring at higher levels and which are not considered to pose an acute hazard are rated 2 through 5, based on the "critical" effect. The critical effect is the effect which occurs at the lowest exposure level, as judged by the threshold level in a toxicity study or, when not

^b A potency slope is a factor which when multiplied by the dose of a carcinogen gives the associated lifetime cancer risk. Potency values represent the theoretical probability of extra cancer cases occurring in the exposed population assuming 70 year lifetime exposure.

^c A unit risk is the estimated probability of a person contracting as a result of constant exposure to an ambient concentration (usually 1 $\mu\text{g}/\text{m}^3$) of a carcinogen over a 70 year lifetime.

available, by a NOAEL^d or LOAEL^e. When a threshold level, NOAEL or LOAEL is not available, a reference exposure level (EPA-IRIS, CAPCOA, OEHHA) or threshold limit value (ACGIH) is used for this level. When not enough information is available to determine which effect occurs at the lowest exposure level, the most commonly cited effect is used as the critical effect.

Overall Toxicity Rating Scale: Levels are measured in $\mu\text{g}/\text{kg}$ or $\mu\text{g}/\text{m}^3$ (single dose or exposure); or $\mu\text{g}/\text{kg}/\text{day}$ or $\mu\text{g}/\text{m}^3/\text{day}$ (chronic exposure).

1. Chemical has severe acute effects, or critical toxic effect of chemicals occurs at levels $< 5 \mu\text{g}$.
2. Critical toxic effect of chemical occurs at levels from 5 - 50 μg .
3. Critical toxic effect of chemical occurs at levels from 50 - 500 μg .
4. Critical toxic effect of chemical occurs at levels from 500 - 5,000 μg .
5. Critical toxic effect of chemical occurs at levels $> 5,000 \mu\text{g}$.

Lethal Dose: Sources: RTECS and Sax. In general, LD₅₀s for inhalation, oral, or skin exposure should be given more weight in assessing the practical hazards of chemical exposure than LD₅₀s based on intraperitoneal, implant, etc., administration.

Toxic endpoints

associated with exposure: Sources: IRIS, RTECS, Sax, CAPCOA, OEHHA, HEAST.

Acute noncancer

reference exposure level: Source: CAPCOA.

Chronic noncancer

reference exposure level: Sources: IRIS, CAPCOA, HEAST and 1993-1994 ACGIH. For EPA reference exposure levels obtained from IRIS, the critical effect on which the level is based is noted, along with information on the confidence of the study. For reference exposure levels obtained from other sources, information on critical effect and confidence level of study is noted as available. ACGIH TLV-TWAs^f have been noted, and in some cases may be the only level analogous to "reference exposure level" available.

^d NOAEL = No observed adverse effect level.

^e LOAEL = Lowest observed adverse effect level.

^f ACGIH TLV-TWA: American Conference of Governmental Industrial Hygienists Threshold Limit Value-Time Weighted Average = The time weighted average airborne concentration of a substance, for a normal 8-hour workday and a 40-hour workweek, to which it is believed that nearly all workers may be repeatedly exposed day after day without adverse health effects.

Critical effect upon which acceptable exposure levels are based/confidence level of critical study: Sources: IRIS, CAPCOA, HEAST, 1991 ACGIH.

Teratogenicity: Sources: IRIS, RTECS, Sax, Lewis.

Hepatotoxicity: Sources: IRIS, RTECS, Sax, Meeks, Gosselin, Zimmerman.

Immunotoxicity: Sources: Bendix Environmental Research, Inc. Immunotoxicology Database, IRIS, CAPCOA, Sax.

Gosselin Toxicity Rating: Source: Gosselin. Gosselin rates chemical toxicity from 1 (low) to 6 (high), based on mortality, not morbidity, making more of a "lethality" rating than a toxicity rating. This rating is usually based on the acute toxicity of a single dose when taken by mouth or gavage.

- 1 - Practically non-toxic. Probable oral lethal dose >15 g/kg.
- 2 - Slightly toxic. Probable oral lethal dose 5-15 g/kg.
- 3 - Moderately toxic. Probable oral lethal dose 0.5-5 g/kg.
- 4 - Very toxic. Probable oral lethal dose 50-500 mg/kg.
- 5 - Extremely toxic. Probable oral lethal dose 5-50 mg/kg.
- 6 - Supertoxic. Probable oral lethal dose <5 mg/kg.

Miscellaneous: Source: Miscellaneous. Other relevant information which does not fit into the above listed categories.

Table 1: Summary of Carcinogenic and Non-Carcinogenic Toxicology

Rating/Toxicity Category	Cd	Dioxin	Fe	Pb	LI	NI	Na	S	H ₂ SO ₄	V	Zn
Overall Carcinogenicity Rating	1	2.5	3	2.5	3	1	3	3	1	3	3
IARC Cancer Rating	1	2B	NR ¹	2B	NR	1 ²	NR	NR	1	NR	NR
EPA Weight-of-Evidence Group	B1	B2	NR	B2	NR	A ³	NR	NR	NR	NR	D
Mutagen	+	++	NR	+	±	+	NR	NR	±	+	± ⁵
Overall Non-Carcinogenic Toxicology Rating	1 (oral)	1 (oral)	5 (inhl.) ⁶	1 (oral)	1 (acute burn hazard)	3 (inhl.)	1 (acute burn hazard)	5 (eye)	1 (acute burn hazard)	3 (inhl.) ⁷	3 (inhl.) ⁴
Teratogen	+	++	NR	+	+	+	+	NR	±	±	+
Liver	X	X	X	X		X		X ⁴			
Immune System	X	X		X		X				X ⁸	X
Cardiovascular/Blood	X		X	X	X	X ⁹					X
Central/Peripheral Nervous Systems				X	X ¹⁰	X ⁹					
Kidney	X	X		X	X	X	X ¹¹				
Respiratory	X	X	X ⁶			X		X	X	X ⁴	X ⁴
Skin		X			X ¹²	X	X ¹²	X (eye)	X	X ⁴	X
Gastro-intestinal		X	X	X							

Mutagen and teratogen ratings: ++ = Effect at less than 10 µg/kg (Potent); + = effect at greater than 10 µg/kg; ± = Single study or inconclusive studies.

Shaded cells indicate critical effect used to rate toxicity.

1. NR = Not rated or no relevant information available. However, iron foundry work is rated in IARC Group 1. Iron oxide rated in IARC Group 3. 2. IARC Group 1 rating for nickel compounds as a group. 3. EPA Weight-of-Evidence Group A rating for nickel refinery dust and nickel subsulfide. 4. Some compounds. 5. Zinc chloride. 6. Iron oxide, measured as Fe. 7. Vanadium pentoxide. 8. Vanadium trioxide and vanadium pentoxide can cause asthma in humans. 9. Nickel salts. 10. Lithium ion. 11. Sodium ion. 12. Metallic form.

Table 2: Cancer Risk and Lethal Dose/Concentration		
Material	Cancers per 10 ⁶ Persons at Dose/Concentration*	LC/LD ₅₀ [†]
Cadmium (Cd)	CAPCOA & OEHA: 4200 at 1 $\mu\text{g}/\text{m}^3$ (inhalation). Calculated from listed inhalation unit risk ($\mu\text{g}/\text{m}^3$) ⁻¹ : 4.2×10^{-3} .	LC ₅₀ 25 mg/m ³ /30 min. (inhalation, rat)
Dioxin (2,3,7,8-Tetra-chlorodibenzo-p-dioxin)	CAPCOA & OEHA: 38 at 1 pg/m^3 (inhalation). Calculated from listed inhalation unit risk ($\mu\text{g}/\text{m}^3$) ⁻¹ : 3.8×10^{-1} . CAPCOA: 133,000 at 1 $\text{pg}/\text{kg}/\text{day}$ (oral). Calculated from listed oral potency value (mg/kg/day) ⁻¹ : 1.33×10^{-5} .	LD ₅₀ 0.6 $\mu\text{g}/\text{kg}$ (oral, guinea pig)
Iron (Fe)	Not a known carcinogen.	LD ₅₀ 20 mg/kg (oral, guinea pig)
Lead (Pb)	Jim Collins, OEHA: 12 at 1 $\mu\text{g}/\text{m}^3$ (inhalation). Calculated from current OEHA inhalation unit risk ($\mu\text{g}/\text{m}^3$) ⁻¹ : 1.2×10^{-3} , not in print. Jim Collins, OEHA: 8,500 at 1 mg/kg/day (oral). Calculated from current OEHA oral potency value (mg/kg/day) ⁻¹ : 8.5×10^{-3} , not in print.	LD ₅₀ 160 mg/kg (oral, pigeon)
Lithium (Li)	Not a known carcinogen.	LD ₅₀ 710 mg/kg (oral, rat)
Nickel (Ni)	OEHA & CAPCOA: 260 at 1 $\mu\text{g}/\text{m}^3$ (inhalation) ^{††} . Calculated from listed inhalation unit risk ($\mu\text{g}/\text{m}^3$) ⁻¹ : 2.6×10^{-4} .	LD ₅₀ 5 mg/kg (oral, guinea pig)
Sodium (Na)	Not a known carcinogen.	LD ₅₀ 4 g/kg (intraperitoneal, mouse)
Sulfur (S)	Not a known carcinogen.	LD ₅₀ 175 mg/kg (oral, rabbit) ^{**}
Sulfuric acid (H ₂ SO ₄)	Known carcinogen. No information on potency available from which to calculate risk.	LC ₅₀ 18 mg/m ³ (inhalation, mouse)
Vanadium (V)	Not a known carcinogen.	LD ₅₀ 23 mg/kg (oral, mouse) [‡]
Zinc (Zn)	Not a known carcinogen.	No LC/LD ₅₀ reported

* Unless otherwise noted, the more conservative Unit Risk or Potency Slope (between OEHA, CAPCOA, and EPA-IRIS) has been used to calculate the cancer risk. Risk values assume continuous exposure over a 70 year lifetime. Values listed as Preliminary have not been used. † LD₅₀s were chosen to be conservative. If no human data were available, or if human data were limited, the most sensitive species for which oral or inhalation data were found was used as the basis for the LC/LD₅₀. If no oral LD₅₀s or inhalation LC₅₀s were available, the most sensitive species for which other exposure data (subcutaneous, intravenous, intraperitoneal, etc.) were found was used as the basis for the LD₅₀. LC/LD₅₀s were used if no LC/LD₅₀s were available. ‡ Vanadium pentoxide. ** Lithium carbonate. †† Nickel and nickel compounds.

CADMIUM CARCINOGENICITY

Overall Carcinogenicity Rating: 1 (IARC 1, EPA B1); human carcinogen.

IARC Cancer Rating: RTECS: IARC Group 1; human carcinogen.

EPA Cancer Weight-of-Evidence Group: IRIS: B1; probable human carcinogen. Basis for classification: Limited evidence from occupational epidemiologic studies of cadmium is consistent across investigators and study populations. There is sufficient evidence of carcinogenicity in rats and mice by inhalation and intramuscular and subcutaneous injection. Seven studies in rats and mice wherein cadmium salts (acetate, sulfate, chloride) were administered orally have shown no evidence of carcinogenic response. (References¹).

Inhalation Cancer Potency Value: CAPCOA & OEHA: Inhalation Unit Risk ($\mu\text{g}/\text{m}^3$)⁻¹: 4.2×10^{-3} .

IRIS: Inhalation Unit Risk: 1.8×10^{-3} per ($\mu\text{g}/\text{m}^3$). "The unit risk should not be used if the air concentration exceeds $6 \mu\text{g}/\text{m}^3$, since above this concentration the unit risk may not be appropriate."

"Risk/Air Concentrations

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
10^{-4} (1 in 10,000)	$6 \times 10^{-2} \mu\text{g}/\text{m}^3$
10^{-5} (1 in 100,000)	$6 \times 10^{-3} \mu\text{g}/\text{m}^3$
10^{-6} (1 in 1,000,000)	$6 \times 10^{-4} \mu\text{g}/\text{m}^3$

Non-Inhalation Cancer Potency Value: NR^g IRIS, NR CAPCOA

HEAST: *Provisional* oral slope comment: "There is inadequate evidence for the carcinogenicity of this compound by the oral route."

Carcinogenic Potential: Sax: Confirmed human carcinogen with experimental carcinogenic, tumorigenic, and neoplastigenic data.

RTECS: TC_{Lo}^h woman: $129 \mu\text{g}/\text{m}^3/20$ Years-constant, inhalation. Tumors of lungs, thorax, respiratory.

^g NR = "Not rated by source", or "no relevant data in source".

^h TC_{Lo}: Toxic Concentration Low - The lowest concentration of a material in air to which humans or animals have been exposed for any given period of time that has been reported to produce a toxic effect.

TD_{Lo}¹ rat: 3372 ug/kg, subcutaneous. Tumorigenic (Carcinogenic by RTECS criteria; Tumors at site of application).

TD_{Lo} rat: 40 mg/kg/4 Weeks, intermittent doses. Tumorigenic/tumors at site of application.

TD¹ rat: 45 mg/kg/4 Weeks, intermittent doses. Tumorigenic (Neoplastic by RTECS criteria, tumors at site of application, musculo-skeletal tumors).

IRIS: "HUMAN: Limited. A 2-fold excess risk of lung cancer was observed in cadmium smelter workers. The cohort consisted of 602 white males who had been employed in production work a minimum of 6 months during the years 1940-1969. The population was followed to the end of 1978. Urine cadmium data available for 261 workers employed after 1960 suggested a highly exposed population. The authors were able to ascertain that the increased lung cancer risk was probably not due to the presence of arsenic or to smoking (Thun, *et al.*, 1985). An evaluation by the Carcinogen Assessment Group of these possible confounding factors has indicated that the assumptions and methods used in accounting for them appear to be valid. As the SMRs observed were low and there is a lack of clear cut evidence of a causal relationship of the cadmium exposure only, this study is considered to supply limited evidence of human carcinogenicity."

"An excess lung cancer risk was also observed in three other studies which were, however, compromised by the presence of other carcinogens (arsenic, smoking) in the exposure or by a small population (Varner, 1983; Sorahan and Waterhouse, 1983; Armstrong and Kazantzis, 1983)."

"Four studies of workers exposed to cadmium dust or fumes provided evidence of a statistically significant positive association with prostate cancer (Kipling and Waterhouse, 1967; Lemen, *et al.*, 1976; Holden, 1980; Sorahan and Waterhouse, 1983), but the total number of cases was small in each study. The Thun, *et al.* (1985) study is an update of an earlier study (Lemen, *et al.*, 1976) and does not show excess prostate cancer risk in these workers. Studies of human ingestion of cadmium are inadequate to assess carcinogenicity."

IRIS: "ANIMAL: Exposure of Wistar rats by inhalation to cadmium as cadmium chloride at concentrations of 12.5, 25 and 50 ug/cu.m for 18 months, with an additional 13-month observation period, resulted in significant increases in lung tumors (Takenaka *et al.*, 1983). Intratracheal instillation of cadmium oxide did not produce lung tumors in Fischer 344 rats but rather mammary tumors in males and tumors at multiple sites in males (Sanders and Mahaffey, 1984). Injection site tumors and distant site tumors (for example, testicular) have been reported by a number of authors as a consequence of intramuscular or subcutaneous administration of cadmium metal and chloride, sulfate, oxide and sulfide compounds of cadmium to rats and mice (U.S. EPA, 1985). Seven studies in rats and mice where cadmium salts (acetate, sulfate, chloride) were administered orally have shown no evidence of a carcinogenic response."

¹ TD_{Lo}: Toxic Dose Low - The lowest dose of a material, administered by any route other than inhalation, over any given period of time and reported to produce a toxic effect.

¹ TD: Toxic Dose - Dose at which a toxic effect is reported to have taken place.

Mutagenic Potential: RTECS: Mouse/embryo: micronucleus test, 6 $\mu\text{mol/L}$.
Hamster/ovary: cytogenetic analysis, 1 $\mu\text{mol/L}$.

IRIS: "Results of mutagenicity tests in bacteria and yeast have been inconclusive. Positive responses have been obtained in mutation assays in Chinese hamster cells (Dom and V79 lines) and in mouse lymphoma cells (Casto, 1976; Ochi and Ohsawa, 1983; Oberly, *et al.*, 1982). "

"Conflicting results have been obtained in assays of chromosomal aberrations in human lymphocytes treated *in vitro* or obtained from exposed workers. Cadmium treatment *in vivo* or *in vitro* appears to interfere with spindle formation and to result in aneuploidy in germ cells of mice and hamsters (Shimada, *et al.*, 1976; Watanabe, *et al.*, 1979; Gilliavod and Leonard, 1975)."

CADMIUM NON-CARCINOGENIC TOXICOLOGY

Overall Non-carcinogenic

Toxicology Rating: 1. Critical effect: Significant proteinuria (kidney) @ 5 µg/kg/chronic exposure, oral.

Lethal Dose: Sax: LC_{Lo}^k human: 39 mg/m³/20 minutes, inhalation.

LD_{Lo}^l man: 15 mg/kg, unknown administration.

LD₅₀^m rat: 225 mg/kg, oral.

LD₅₀ rat: 4 mg/kg, intraperitoneal.

LD₅₀ rat: 9 mg/kg, subcutaneous.

LD₅₀ rat: 1.8 mg/kg, intravenous.

LC₅₀ⁿ rat: 25 mg/m³/30 minutes, inhalation.

LD₅₀ mouse: 890 mg/kg, oral.

LC_{Lo} mouse: 170 mg/m³, inhalation.

(List truncated.)

RTECS: Cited same studies as Sax, above, plus:

LD₅₀ mouse: 5.7 mg/kg, intraperitoneal.

LD_{Lo} rabbit: 70 mg/kg, oral.

LD_{Lo} rabbit: 5 mg/kg, oral.

Toxic endpoints

associated with exposure: Gosselin: Acute inhalation of dust or fumes - pulmonary edema, necrosis of the pulmonary epithelium...following absorption, Cd accumulates in the liver and kidneys...renal injuries are common among workers in cadmium industries whether the exposure is acute or chronic over many years...acute massive exposures may result in acute renal necrosis...soluble cadmium salts given parenterally to rats, mice, or calves induced a temporary castration phenomenon, which progressed with larger doses to irreversible damage to the seminiferous tubules, necrosis of the germinal epithelium, and complete loss of fertility...damage to seminiferous tubules has not been observed in chronically exposed men...*clearly, the major target organs for parenteral or oral cadmium are the kidney and the liver.*

^k LC_{Lo}: Lethal Concentration Low - The lowest concentration of a material in air (other than LC₅₀) which has been reported to have caused death in humans or animals.

^l LD_{Lo}: Lethal Dose Low - The lowest dose (other than LD₅₀) of a material, administered by any route other than inhalation, over any given period of time, in one or more divided portions and reported to have caused death in humans or animals.

^m LD₅₀: Lethal Dose Fifty - A calculated dose of a material which is expected to cause the death of 50% of an entire defined experimental animal population.

ⁿ LC₅₀: Lethal Concentration Fifty - A calculated concentration of a material in air, exposure to which for a specified length of time is expected to cause the death of 50% of an entire defined experimental animal population.

Sax: Human poison by inhalation and possibly other routes. Experimental poison by ingestion, inhalation, and other routes. Reproductive effects.

CAPCOA: Kidney, respiratory system.

RTECS: Proteinuria (kidney, ureter, bladder), cardiac, vascular, lungs and respiratory

NIOSH: Respiratory system, kidneys, prostate, blood (cadmium dust).

Respiratory system, kidneys, blood (cadmium fume).

Acute noncancer

reference exposure level^o: NR CAPCOA

Chronic noncancer

reference exposure level: IRIS: Oral reference dose (oral RfD), 5×10^{-3} mg/kg/day (water). 1.0×10^{-3} mg/kg/day (food).

CAPCOA: Oral reference exposure level, 1.0×10^{-3} mg/kg/day.

CAPCOA: Inhalation reference exposure level, 3.5×10^{-6} $\mu\text{g}/\text{m}^3$. (This value was calculated in CAPCOA from the IRIS oral acceptable exposure level (mg/kg/day) by assuming a 70 kg person breathes 20 m^3 per day and equal absorption occurs by the inhalation and oral routes.)

**Critical effect upon which
acceptable exposure levels
are based/confidence level**

of critical study: IRIS: Critical effect for IRIS RfD is significant proteinuria^p. NOAEL^q (water) 0.005 mg/kg/day. NOAEL (food) 0.01 mg/kg/day.

"Critical study used in setting RfD: The choice of NOAEL does not reflect the information from any single study. Rather, it reflects the data obtained from many studies on the toxicity of cadmium in both humans and animals. These data also permit calculation of pharmacokinetic parameters of cadmium absorption, distribution, metabolism and elimination. All of this information considered together gives high confidence in the data base. High confidence in either RfD follows as well."

^o Noncancer reference exposure levels are concentrations ($\mu\text{g}/\text{m}^3$) or doses (mg/kg/day) at or below which no adverse health effects (not including cancer) are anticipated.

^p Proteinuria = the presence of an excess of serum proteins in the urine.

^q NOAEL = no observed adverse effect level.

"Oral RfD Confidence:
Study -- Not applicable.
Data Base -- High
RfD -- High"

"An uncertainty factor of 10 is used to account for intrahuman variability to the toxicity of this chemical in the absence of specific data on sensitive individuals."

Teratogenicity: Sax: "Experimental teratogenic effects." TD_{Lo} rat: 21.5 mg/kg, multigenerational study, inhalation. Teratogenic effects.

RTECS: TD_{Lo} rat: 23 mg/kg (1-22 days preg.), oral. Specific developmental abnormalities (Blood and lymphatic systems).

TD_{Lo} rat: 1.25 mg/kg (14 days preg.), intravenous. Specific developmental abnormalities (Body wall; Urogenital system).

TD_{Lo} rat: 1.25 mg/kg (9 days preg.), intravenous. Specific developmental abnormalities (Central nervous system; eye; ear).

TD_{Lo} mouse: 1.69 mg/kg (7 days preg.), intraperitoneal. Specific developmental abnormalities (Central nervous system).

TD_{Lo} hamster: 2 mg/kg (8 days preg.), parenteral. Specific developmental abnormalities (Craniofacial).

(List does not include RTECS TDs for "effects on embryo or fetus", "effects on newborn", or "paternal effects".)

Lewis: No studies besides those cited by RTECS.

IRIS: "FIFRA Review: Summary of regulatory action -- All uses of cadmium pesticides canceled. Criteria of concern: oncogenicity, mutagenicity, teratogenicity, and fetotoxicity."

Hepatotoxicity: Zimmerman: "Pollution of the environment by metals or metalloids would at present appear to pose little threat of hepatotoxicity. Cadmium, however, is a true experimental hepatotoxin, able to produce hepatic degeneration, necrosis, and cirrhosis in experimental animals. Both lead and cadmium can enhance the hepatic injury produced in experimental animals by endotoxin. There is no evidence, however, that the environmental exposure to any of these metals can be implicated in human hepatic injury. Nevertheless, increased metallic stores in human tissues...may prove to have a bearing in cryptogenic liver disease."

Gosselin: Following absorption, cadmium accumulates in the liver and kidneys.

NR RTECS, NR IRIS, NR Sax, NR Meeks

Immunotoxicity: "Examples of chemicals affecting immunologic suppression or enhancement in rodents and/or humans include...cadmium"² Inhibit natural killer (NK) cell-mediated toxicity of human peripheral blood lymphocytes.³ In 12-hr assay, cadmium-

treated animals had significantly lower NK cell activity than controls. In 4-hr assay, a similar trend was observed.⁴

Gosselin Toxicity Rating: "5(?). Inhaled as a dust or aerosol, cadmium and its salts (even the relatively insoluble oxide) probably have a toxicity rating of 6 in man, with death from fatal pulmonary injury. When swallowed, these salts (i.e., chloride, oxide, sulfate) are much less lethal, in part because they induce vomiting and are not retained. Although as little as 10 mg. of cadmium salts has often produced severe toxic symptoms when ingested, a toxicity rating of 5 is probably a reasonable estimate of cadmium's lethality by the oral route...Death probably requires several hundred mg by the oral route"

DIOXIN CARCINOGENICITY

Overall Carcinogenicity Rating: 2.5 (IARC 2B, EPA B2); possible/probable human carcinogen. Data available in a review draft of a report⁵ not yet formally released by the EPA indicate that a rating of 2 (IARC 2A, EPA B1) may be more appropriate.

IARC Cancer Rating: IARC Supplement 7: IARC Group 2B; possible human carcinogen.

EPA Cancer Weight-of-Evidence Group: EPA and HEAST: B2; probable human carcinogen.

Inhalation Cancer Potency Value: CAPCOA & OEHHHA: Inhalation unit risk ($\mu\text{g}/\text{m}^3$)⁻¹: 3.8×10^{-1} (chlorinated dibenzo-p-dioxins, as 2,3,7,8-equivalents).

HEAST: *Provisional* inhalation slope factor ($\text{mg}/\text{kg}/\text{day}$)⁻¹: 1.5×10^{-5} . *Provisional* inhalation unit risk (pg/m^3)⁻¹: 3.3×10^{-5} ("Under review, number subject to change. Based on route to route extrapolation. An absorption factor of 75% is used to calculate the unit risk from the slope factor.").

NR IRIS

Non-Inhalation Cancer Potency Value: EPA: Cancer Slope^a ($\text{mg}/\text{kg}/\text{day}$)⁻¹: 1.56×10^{-5} .

CAPCOA: Oral cancer potency value ($\text{mg}/\text{kg}/\text{day}$)⁻¹: 1.33×10^{-5} (chlorinated dibenzo-p-dioxins, as 2,3,7,8-equivalents)

OEHHHA: Oral potency slope ($\text{mg}/\text{kg}/\text{day}$)⁻¹: 1.3×10^{-5} .

HEAST: *Provisional* oral slope factor ($\text{mg}/\text{kg}/\text{day}$)⁻¹: 1.5×10^{-5} ("Under review, subject to change."). *Provisional* oral unit risk ($\mu\text{g}/\text{L}$)⁻¹: 4.5×10^{-6} .

EPA2: Cancer potency ($0.01 \text{ pg}/\text{kg}/\text{day}$)⁻¹: 1×10^{-6} (TCDD and related compounds). The EPA source for this value is a "Review Draft"; this value is not yet final.)

NR IRIS

Carcinogenic Potential: RTECS: TD_{Lo} rat: $52 \mu\text{g}/\text{kg}/2$ years-intermittent, oral. Tumorigenic (Carcinogenic by RTECS criteria; Tumors of liver and thyroid).
 TD_{Lo} mouse: $52 \mu\text{g}/\text{kg}/2$ years-intermittent, oral. Tumorigenic (Carcinogenic by RTECS criteria; Tumors of liver and thyroid).

¹ 2,3,7,8-Tetrachloro-dibenzo-p-dioxin, generally considered the most toxic dioxin, is used as the basis for this assessment.

^a 95% upper-bound slope based on animal oral studies.

TD_{Lo} mouse: 62 µg/kg/2 years-intermittent, skin. Tumorigenic (Carcinogenic by RTECS criteria; Tumors of skin and appendages).

TD rat: 73 µg/kg/2 years-continuous, oral. Tumorigenic (Carcinogenic by RTECS criteria; Tumors of liver).

TD mouse: 36 µg/kg/52 weeks-intermittent, oral. Tumorigenic (Neoplastic by RTECS criteria; Tumors of liver and lungs).

NR IRIS

Mutagenic Potential: RTECS: *S. typhimurium*: Mutation in microorganisms, 2 mg/L (+S9).

E. coli: Mutation in microorganisms, 2 mg/L (-S9).

E. coli: Phage inhibition capacity, 500 µg/L.

S. cerevisiae: Mutation in microorganisms, 10 mg/L (-S9).

S. cerevisiae: Mutation in microorganisms, 2 mg/L (+S9).

S. cerevisiae: Gene conversion and mitotic recombination, 10 mg/L.

Human, cell types: Unscheduled DNA synthesis, 100 pmol/L.

Human, cell types: DNA inhibition, 10 nmol/L.

Rat/oral: DNA damage, 100 µg/kg.

Rat/intravenous: Unscheduled DNA synthesis, 4 µg/kg.

Rat/oral: Unscheduled DNA synthesis, 5 µg/kg.

Rat/intraperitoneal: DNA inhibition, 10 µg/kg.

Rat/oral: DNA inhibition, 200 µg/kg.

Rat/oral: Cytogenetic analysis, 100 µg/kg.

Rat/intraperitoneal: Cytogenetic analysis, 10 mg/kg.

Mouse, fibroblast: Morphological transformation, 200 nmol/L.

Mouse, cell types: Test systems (other), 1 nmol/L.

Mouse/oral: Unscheduled DNA synthesis, 800 pmol/kg.

Mouse/intraperitoneal: DNA inhibition, 400 µg/kg.

Mouse/oral: Test systems (other), 50 µg/kg.

Mouse/oral: Cytogenetic analysis, 100 µg/kg.

Mouse/intraperitoneal: Cytogenetic analysis, 10 µg/kg.

Mouse, lymphocyte: Gene mutation in mammalian cells, 50 mg/L.

Mouse/*S. cerevisiae*: Host-mediated assay, 25 µg/kg.

Sax: "Human mutation data reported."

NR IRIS

DIOXIN NON-CARCINOGENIC TOXICOLOGY

Overall Non-carcinogenic

Toxicology Rating: 1. Critical effect: Teratogenesis @ 1 µg/kg, oral, mouse.

Lethal Dose: RTECS: LD₅₀ rat: 20 µg/kg, oral.

LD₅₀ mouse: 114 µg/kg, oral.

LD_{Lo} mouse: 80 µg/kg, skin.

LD₅₀ dog: 1 µg/kg, oral.

LD₅₀ monkey: 2 µg/kg, oral.

LD₅₀ rabbit: 115 µg/kg, oral.

LD₅₀ rabbit: 275 µg/kg, skin.

LD₅₀ guinea pig: 500 ng/kg, oral.

LD₅₀ hamster: 1157 µg/kg, oral.

LD_{Lo} chicken: 25 µg/kg, oral.

LD₅₀ frog: 1 mg/kg, oral.

Gosselin: "Acute oral LD₅₀s for TCDD range from 0.6 µg/kg in male guinea pigs, to 115 µg/kg for rabbits of mixed sex."

Toxic endpoints

associated with exposure: RTECS: Human skin (allergic dermatitis after topical application; 107 µg/kg.); liver (hepatitis, hepatocellular necrosis, change in liver weight); endocrine (changes in thymus and spleen weights); changes in kidney, ureter, bladder; respiratory (dyspnea).

CAPCOA: Immune system, gastrointestinal or liver, reproductive system, skin.

EPA2: A large data base "suggests that exposure to dioxin results in a broad spectrum of biochemical and biological effects in animals and, based on limited data, some of these effects occur in humans."

Sax: Skin (allergic dermatitis by skin contact); poison by ingestion; eye irritant; death in exposed rats by hepatic cell necrosis; acute and subacute exposure result in wasting, hepatic necrosis, thymic atrophy, hemorrhage, lymphoid depletion, chloracne.

NR IRIS, NR NIOSH

Acute noncancer

reference exposure level: NR CAPCOA

Chronic noncancer

reference exposure level: CAPCOA: Oral reference exposure level (mg/kg/day): 1.0×10^{-9} (chlorinated dibenzo-p-dioxins, as 2,3,7,8-equivalents). Inhalation reference exposure level (µg/m³): 3.5×10^{-6} (chlorinated dibenzo-p-dioxins, as 2,3,7,8-equivalents) (This value was

calculated in CAPCOA from oral acceptable exposure level (mg/kg/day) by assuming a 70 kg person breathes 20 m³ per day and equal absorption occurs by the inhalation and oral routes.)

NR IRIS, NR HEAST, NR 1993-1994 ACGIH

**Critical effect upon which
acceptable exposure levels
are based/confidence level
of critical study: NR CAPCOA**

Teratogenicity: RTECS: TD_{Lo} rat: 1500 ng/kg (1-3 day preg.), oral. Specific developmental abnormalities (urogenital system).

TD_{Lo} rat: 1250 ng/kg (6-15 day preg.), oral. Fetal death; fetotoxicity. Specific developmental abnormalities (Homeostasis).

TD_{Lo} rat: 1270 ng/kg, oral, multi generations. Specific developmental abnormalities (Blood and lymphatic systems). Effects on newborn (growth statistics).

TD_{Lo} rat: 127 ng/kg, oral, multi generations. Specific developmental abnormalities (urogenital system). Effects on newborn (Live birth index; weaning and lactation index).

TD_{Lo} rat: 5 mg/kg (6-15 days preg.), subcutaneous. Specific developmental abnormalities (urogenital system).

TD_{Lo} mouse: 1 µg/kg (10 days preg.), oral. Specific developmental abnormalities (urogenital system).

TD_{Lo} mouse: 9 µg/kg (12 days preg.), oral. Specific developmental abnormalities (craniofacial).

TD_{Lo} mouse: 13.5 g/kg (6-14 days preg.), oral. Specific developmental abnormalities (endocrine system).

TD_{Lo} mouse: 12 µg/kg (10-13 days preg.), oral. Effects on fertility (post-implantation mortality). Effects on embryo of fetus (fetal death).

TD_{Lo} mouse: 250 µg/kg (7-16 days preg.), subcutaneous. Specific developmental abnormalities (Craniofacial; Musculoskeletal system; Urogenital system)

TD_{Lo} mouse: 30 µg/kg (10 days preg.), subcutaneous. Specific developmental abnormalities (craniofacial).

(list truncated)

Lewis: TD_{Lo} mouse: 235 µg/kg (28 day preg.), oral. Specific developmental abnormalities (immune and reticuloendothelial system).

TD_{Lo} mouse: 23 µg/kg (11 days post), oral. Specific developmental abnormalities (craniofacial, including nose and tongue).

NR IRIS

Hepatotoxicity: RTECS: Multiple dose toxicity study: TD_{Lo} rat: 65 mg/kg/13 weeks-intermittent, oral. Changes in weights of liver & thymus.

Multiple dose toxicity study: TD_{Lo} rat: 7300 mg/kg/2 year-continuous, oral. Hepatitis: hepatocellular necrosis, zonal; changes in liver weight.

Multiple dose toxicity study: TD_{Lo} rat: 16 µg/kg/16 weeks-intermittent, oral. Changes in liver.

Multiple dose toxicity study: TD_{Lo} mouse: 588 µg/kg/14 days-intermittent, oral. Changes in liver, spleen and thymus weights.

Multiple dose toxicity study: TD_{Lo} mouse: 97 µg/kg/13 weeks-intermittent, skin. Hepatitis: hepatocellular necrosis, diffuse.

Multiple dose toxicity study: TD_{Lo} guinea pig: 441 ng/kg/90 days-continuous, oral. Changes in liver and thymus weights.

Meeks: "2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) [has] been shown to produce hepatic uroporphyrin not only in experimental animals, but also in humans."

Zimmerman: "[2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)] is an enormously potent hepatotoxin that can lead to centrilobular necrosis and toxic porphyria."

Sax: Death in exposed rats by hepatic cell necrosis.

NR IRIS

Immunotoxicity: "Dioxin is highly immunotoxic in the mouse...*In vitro* studies have suggested that mouse, monkey, and human lymphocytes are responsive to dioxin effects. Recent studies have demonstrated, however, that the rat is relatively resistant to the immunosuppressive effects of TCDD."⁶ Causes immunosuppression.⁷ "Examples of chemicals affecting immunologic suppression or enhancement in rodents and/or humans include... (2,3,7,8-tetrachlorodibenzo(p)dioxin..."⁸

Gosselin Toxicity Rating: "6. For its molecular weight, TCDD may be the most toxic and potent teratogen known to man."

IRON CARCINOGENICITY

Overall Carcinogenicity Rating: 3; not classifiable. However, iron foundry work is rated in IARC Group 1.

IARC Cancer Rating: IARC Supplement 7: Iron not rated by IARC. However, iron foundry work is rated in Group 1.

EPA Cancer Weight-of-Evidence Group: NR EPA, NR IRIS

Inhalation Cancer Potency Value: NR IRIS, NR CAPCOA, NR OEHHA

Non-Inhalation Cancer Potency Value: NR IRIS, NR CAPCOA, NR OEHHA

Carcinogenic Potential: RTECS: TD_{Lo} rat: 450 mg/kg/15 weeks, intermittent doses, intra-tracheal. Tumorigenic (equivocal tumorigenic agent by RTECS criteria. Tumors of lungs, thorax, respiratory).

Sax: Cites same study as above.

NR IRIS

Mutagenic Potential: NR IRIS, NR RTECS, NR Sax

IRON NON-CARCINOGENIC TOXICOLOGY

Overall Non-carcinogenic

Toxicology Rating: 5. Critical effect: Benign pneumoconiosis (respiratory) @ 15,000 µg/m³/chronic occupational exposure, inhalation, human.

Lethal Dose: Sax: LD₅₀ rat: 30 g/kg, oral.
LD_{Lo} rabbit: 20 mg/kg, intraperitoneal.

RTECS: LD₅₀ guinea pig: 20 mg/kg, oral.

Toxic endpoints

associated with exposure: RTECS: TD_{Lo} child: 77 mg/kg, oral. Gastrointestinal (nausea, vomiting), behavioral (irritability), blood (normocytic anemia).

Sax: Poison by intraperitoneal route. Iron is potentially toxic in all forms and by all routes of exposure. The inhalation of large amounts of iron dust results in iron pneumoconiosis. Chronic exposure can result in pathological deposition of iron in the body tissues, the symptoms of which are fibrosis of the pancreas, diabetes mellitus, and liver cirrhosis.

NIOSH: Respiratory system (Iron oxide dust and fume, as Fe).

NR CAPCOA

Acute noncancer

reference exposure level: NR CAPCOA

Chronic noncancer

reference exposure level: HEAST: "Data inadequate for quantitative risk assessment."

1993-1994 ACGIH: Not rated as iron. However, Iron oxide fume (welding fume), as Fe, TLV-TWA = 5.0 mg/m³. Iron salts (soluble), as Fe, TLV-TWA = 1.0 mg/m³.

NR CAPCOA, NR IRIS

**Critical effect upon which
acceptable exposure levels
are based/confidence level**

of critical study: 1991 ACGIH: TLV-TWA for iron oxide fume or dust based on critical effect of benign pneumoconiosis. "No studies were reported that would permit a correlation between exposure level and the occurrence of x-ray changes in the lungs. Accordingly, a TLV-TWA of 5.0 mg/m³, measured as Fe, is recommended to minimize the potential for development of x-ray changes in the lung on long-term exposure."

"TLV-TWA for iron salts (soluble), as Fe, is recommended to reduce the likelihood of respiratory irritation and skin irritation from exposures to aerosols and mists of soluble iron salts."

NR IRIS

Teratogenicity: NR IRIS, NR Sax, NR RTECS, NR Lewis

Hepatotoxicity: Sax: Liver cirrhosis can be a symptom of chronic iron exposure due to pathological deposition of iron in body tissues.

Zimmerman: "Acute hepatic necrosis caused by accidental ingestion of large amount of ferrous sulfate has been reported in children."

Meeks: Excessive hemosiderin [an iron containing pigment] can be deposited in the hepatocytes after excessive dietary ingestion of iron and in patients who ingest excessive iron supplements. "Under certain conditions, iron can act as a hepatotoxin, and it plays a catalytic role in the hepatotoxicity of a number of xenobiotics."

NR IRIS, NR RTECS

Immunotoxicity: NR IRIS

Gosselin Toxicity Rating: Iron not rated. Iron salts rated 3.

Miscellaneous: Elemental iron is far less toxic than soluble iron salts.

LEAD CARCINOGENICITY

Overall Carcinogenicity Rating: 2.5 (IARC 2B, EPA B2); possible/probable human carcinogen.

IARC Cancer Rating: IARC Supplement 7: IARC Group 2B; possible human carcinogen (lead & inorganic lead compounds). IARC Group 3; not classifiable as to its carcinogenicity to humans (organolead compounds).

EPA Cancer Weight-of-Evidence Group: IRIS: B2; probable human carcinogen. Basis for classification: Sufficient animal evidence. Ten rat bioassays and one mouse assay have shown statistically significant increases in renal tumors with dietary and subcutaneous exposure to several soluble lead salts. Animal assays provide reproducible results in several laboratories, in multiple rat strains with some evidence of multiple tumor sites. Short term studies show that lead affects gene expression. Human evidence is inadequate. (References⁹).

Inhalation Cancer Potency Value: CAPCOA: Unit Risk ($\mu\text{g}/\text{m}^3$)⁻¹: 8.0×10^{-5} (lead compounds) *Preliminary* Cancer Potency Value for Air Toxics "Hot Spots" Act.

Jim Collins, OEHA: Unit Risk ($\mu\text{g}/\text{m}^3$)⁻¹: 1.2×10^{-5} .

IRIS: "Studies of inhalation exposure have not been located in the literature."

NR OEHA

Non-Inhalation Cancer Potency Value: IRIS: "Quantifying lead's cancer risk involves many uncertainties, some of which may be unique to lead. Age, health, nutritional state, body burden, and exposure duration influence the absorption, release, and excretion of lead. In addition, current knowledge of lead pharmacokinetics indicates that an estimate derived by standard procedures would not truly describe the potential risk. Thus, the Carcinogen Assessment Group recommends that a numerical estimate not be used."

Jim Collins, OEHA: Oral potency slope, NR CAPCOA, NR IRIS, but Jim Collins, OEHA, says "Multi pathway ($\text{mg}/\text{kg}/\text{day}$)⁻¹ (non-inhalation): 8.5×10^{-3} " is the current value used by OEHA, although not in CAPCOA.

Carcinogenic Potential: NR RTECS

Sax: "Suspected carcinogen."

IRIS: "HUMAN: Inadequate. There are four epidemiologic studies of occupational cohorts exposed to lead and lead compounds. Two studies (Dingwall-Fordyce and Lane, 1963; Nelson et al., 1982) did not find any association between exposure and cancer mortality. Selevan et al. (1985), in their retrospective cohort mortality study of primary lead smelter

workers, found a slight decrease in the total cancer mortality (SMR=95). Apparent excesses were observed for respiratory cancer (SMR=111, obs=41, $p>0.05$) and kidney cancer (SMR=204, obs=6, $p>0.05$). Cooper and Gaffey (1975) and Cooper (1985 update) performed a cohort mortality study of battery plant workers and lead smelter workers. They found statistically significant excesses for total cancer mortality (SMR=113, obs=344), stomach cancer (SMR=168, obs=34), and lung cancer (SMR=124, obs=109) in the battery plant workers. Although similar excesses were observed in the smelter workers, they were not statistically significant. Cooper and Gaffey (1975) felt it was possible that individual subjects were monitored primarily on the basis of obvious signs of lead exposure, while others who showed no symptoms of lead poisoning were not monitored."

"All of the available studies lacked quantitative exposure information, as well as information on the possible contribution from smoking. All studies also included exposures to other metals such as arsenic, cadmium, and zinc for which no adjustment was done. The cancer excesses observed in the lung and stomach were relatively small (<200). There was no consistency of site among the various studies, and no study showed any dose-response relationship. Thus, the available human evidence is considered to be inadequate to refute or demonstrate any potential carcinogenicity for humans from lead exposure."

IRIS: "ANIMAL: Sufficient. The carcinogenic potential of **lead salts** (primarily phosphates and acetates) administered via the oral route or by injection has been demonstrated in rats and mice by more than 10 investigators. The most characteristic cancer response is bilateral renal carcinoma. Rats given **lead acetate** or **subacetate** orally have developed gliomas, and lead subacetate also produced lung adenomas in mice after i.p. administration. Most of these investigations found a carcinogenic response only at the highest dose. The lead compounds tested in animals are almost all soluble salts. **Metallic lead, lead oxide and lead tetraalkyls** have not been tested adequately. Studies of inhalation exposure have not been located in the literature. "

"Azar et al. (1973) administered 10, 50, 100, and 500 ppm **lead as lead acetate** in dietary concentrations to 50 rats/sex/group for 2 years. Control rats (100/sex) received the basal laboratory diet. In a second 2-year feeding study, 20 rats/group were given diets containing 0, 1000, and 2000 ppm lead as lead acetate. No renal tumors were reported in the control groups or in treated animals of either sex receiving 10 to 100 ppm. Male rats fed 500, 1000, and 2000 ppm lead acetate had an increased renal tumor incidence of 5/50, 10/20, and 16/20, while 7/20 females in the 2000-ppm group developed renal tumors."

"The Azar et al. (1973) study is limited by the lack of experimental detail. The possibility of environmental contamination from lead in the air or drinking water was not mentioned. The strains of rats used were not specified in the study, but the Health Effects Assessment for Lead (U.S. EPA, 1984) indicates the rats were Wistar strain. The weight gain at 1000 and 2000 ppm was reported to be depressed, but details were not given."

"Kasprzak et al. (1985), in investigating the interaction of dietary calcium on lead carcinogenicity, fed 1% **lead subacetate** (8500 ppm Pb) to male Sprague-Dawley rats in

the diet for 79 weeks. Of the rats surviving (29/30) in this treatment group beyond 58 weeks, 44.8% had renal tumors. Four rats had adenocarcinomas; the remaining nine had adenomas. Bilateral tumors were noted. No renal tumors were noted among the controls."

"As part of a study to determine interactions between sodium nitrite, ethyl urea and lead, male Sprague-Dawley rats were given **lead acetate** in their drinking water for 76 weeks (Koller et al., 1986). The concentration of lead was 2600 ppm. No kidney tumors were detected among the 10 control rats. Thirteen of 16 (81%) lead-treated rats had renal tubular carcinoma; three tumors were detected at 72 weeks and the remainder detected at the termination of the study."

"Van Esch and Kroes (1969) fed basic **lead acetate** at 0, 0.1%, and 1.0% in the diet to 25 Swiss mice/sex/group for 2 years. No renal tumors developed in the control group, but 6/25 male mice of 0.1% basic lead acetate group had renal tumors (adenomas and carcinomas combined). In the 1.0% group, one female had a renal tumor. The authors thought that the low incidence in the 1.0% group was due to early mortality."

"Hamsters given **lead subacetate** at 0.5% and 1% in the diet had no significant renal tumor response (Van Esch and Kroes, 1969)."

Mutagenic Potential: Sax: "Human mutation data reported"

RTECS: Human/unreported route: cytogenic analysis, 50 $\mu\text{g}/\text{m}^3$.

Rat/inhalation: cytogenic analysis, 23 $\mu\text{g}/\text{m}^3$ /16 weeks.

Monkey/oral: cytogenic analysis, 42 mg/kg/30 weeks.

IRIS: "**Lead acetate** induces cell transformation in Syrian hamster embryo cells (DiPaolo, et al., 1978) and also enhances the incidence of simian adenovirus induction. **Lead oxide** showed similar enhanced adenovirus induction (Casto, et al., 1979)."

"Under certain conditions lead compounds are capable of inducing chromosomal aberrations *in vivo* and in tissue cultures. Grandjean, et al. (1983) showed a relationship between SCE and lead exposure in exposed workers. Lead has been shown, in a number of DNA structure and function assays, to affect the molecular processes associated with the regulation of gene expression (U.S. EPA, 1986)."

LEAD NON-CARCINOGENIC TOXICOLOGY

Overall Non-carcinogenic

Toxicology Rating: 1. Critical effect: Nervous system effects in children at concentrations so low as to be essentially without threshold.

Lethal Dose: Sax: LD₅₀ rat: 1000 mg/kg, intraperitoneal.

LD₅₀ pigeon: 160 mg/kg, oral.

RTECS: Cited same studies as Sax, above.

Toxic endpoints

associated with exposure: CAPCOA: (lead and compounds) cardiovascular or blood system, central or peripheral nervous system, immune system, kidney, reproductive system including teratogenic and developmental effects.

Sax: Major organ systems affected are the blood system, nervous system, and kidneys. Poison by ingestion. Human systemic effects by inhalation or ingestion. Impairment of immune system.

RTECS: Peripheral nervous system, gastrointestinal, liver, reproductive system, brain.

NIOSH: Gastrointestinal tract, central nervous system, kidneys, blood, gingival tissue.

Gosselin: Blood, deposition of lead in bone tissue, irritation of alimentary tract after ingestion, peripheral neuritis.

Acute noncancer

reference exposure level: NR CAPCOA

Chronic noncancer

reference exposure level: CAPCOA: Inhalation: $1.5 \times 10^{-6} \mu\text{g}/\text{m}^3$ for lead and compounds (based on conversion from the California Ambient Air Quality Standard - CAAQS).

CAPCOA: Oral reference exposure level: $4.3 \times 10^{-4} \text{ mg}/\text{kg}/\text{day}$ (based on conversion from the California Ambient Air Quality Standard - CAAQS)

IRIS: Oral RfD: It appears that [some toxic effects of lead], particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold. The Agency's [EPA] Work Group discussed inorganic lead (and lead compounds) at two meetings (07/08/85 and 07/22/85) and considered it inappropriate to develop an RfD for inorganic lead.

HEAST: National Ambient Air Quality Standard (NAAQS): $1.5 \mu\text{g}/\text{m}^3$; quarterly averaging time.

1993-1994 ACGIH: Lead, inorganic compounds, dusts and fumes, as Pb, TLV-TWA = $0.15 \text{ mg}/\text{m}^3$. Lead, elemental and inorganic compounds, as Pb = $0.05 \text{ mg}/\text{m}^3$ (on notice of intended changes for 1993-1994).

Critical effect upon which acceptable exposure levels are based/confidence level of critical study: NR IRIS, see above.

1991 ACGIH: Regarding the TLV-TWA for inorganic lead compounds, dusts and fumes, as Pb, of 0.15 mg/m³: new data on the reported health effects from exposure to lead have appeared since this TLV was adopted. The recommended change (TLV-TWA = 0.05 mg/m³) is based on the critical effect of developmental consequences of low-level prenatal exposure to lead.

Teratogenicity: Gosselin: Lead is clearly teratogenic in laboratory animals. It is not clear with certainty whether or not it is teratogenic in humans.

Sax: "An experimental teratogen". LD₅₀ mouse: 4800 mg/kg (female 1-16 days post), oral. Teratogenic effects.

RTECS: TD₅₀ rat: 1100 mg/kg (1-22 days preg.), oral. Specific developmental abnormalities (blood and lymphatic system).

TD₅₀ rat: 10 mg/m³/24 hours (1-21 days preg.), inhalation. Specific developmental abnormalities (blood and lymphatic system).

(List does not include RTECS TDs for "effects on embryo or fetus", "effects on newborn", "effects on fertility".)

Lewis: No studies besides those cited by RTECS.

NR IRIS

Hepatotoxicity: Gosselin: "Hepatitis and hepatic failure, together with more common manifestations of lead poisoning, were prominent in a series of cases involving lead and opium pills which were crushed, suspended in water, and injected intravenously. Although hepatotoxicity in these cases was ascribed to the bizarre route of exposure, liver damage has also occurred following a single massive ingestion of red lead (lead tetroxide)."

Sax: "Lead produces a damaging effect on the organs or tissues with which it comes in contact. No specific lesion is produced. Autopsies in deaths attributable to lead poisoning and experimental work on animals have shown pathological lesions of the kidneys, liver, male gonads, nervous system, blood vessels and other tissues. None of these changes, however, have been found consistently."

Zimmerman: "While [lead, cadmium and mercury] are not ordinarily considered to be hepatotoxic, all can produce hepatic injury in experimental animals. That produced by mercury and lead is slight."

Meeks: "Generalized enlargement of the liver can occur following lead injection."

Immunotoxicity: "Examples of chemicals affecting immunologic suppression or enhancement in rodents and/or humans include...lead..."¹⁰ Workers with lead levels below 60 ug/dc of lead have significantly reduced immune function, as judged by chemotaxis and nitroblue tetrazolium reduction.¹¹ Workers with blood Pb >25 ug/dc have immune dysfunction with significant reduction in CD3+ and CD4+ cells, impaired T-cell response to mitogens, possible impaired feedback loop regulating T-cell dependent functions and cell-to-cell cooperation; marked deficiency in MLC responses.¹² Decreased chemotaxis and phagocytosis in human polymorphonuclear leukocytes.^{13,14} Alters ability of macrophages to present antigen by enhancing the autologous mixed lymphocyte reaction; known to alter various parameters of immune function such as host resistance and antibody formation.¹⁵ Increases IgM production by T-cells in mouse.¹⁶ Suppresses human humoral responsiveness and decreases IgG production.¹⁷ Causes increased susceptibility to disease, kidney tubular cancer, decreased antibody production in animals.¹⁸ Thymic weights significantly decreased; suppression of responsiveness of lymphocytes to mitogen stimulation; reduced delayed hypersensitivity responsiveness; chronic low-level Pb exposure suppresses cell-mediated immune function.¹⁹ Alters ability of macrophages to present antigen.²⁰

CAPCOA: Immune system as target organ.

Gosselin Toxicity Rating: Elemental lead not rated. Most lead compounds lie in toxicity class 3 or 4...Lead is poisonous in all forms. It is one of the most hazardous of the toxic metals because the poison is cumulative and the toxic effects are many and severe.

LITHIUM CARCINOGENICITY

Overall Carcinogenicity Rating: 3; not classifiable.

IARC Cancer Rating: NR IARC

EPA Cancer Weight-of-Evidence Group: NR EPA, NR IRIS

Inhalation Cancer Potency Value: NR CAPCOA, NR OEHHA, NR IRIS

Non-Inhalation Cancer Potency Value: NR CAPCOA, NR OEHHA, NR IRIS

Carcinogenic Potential: NR RTECS, NR IRIS, NR Sax

Mutagenic Potential: NR IRIS, NR Sax

RTECS: Human/unreported route: cytogenic analysis, 714 mg/kg/8 weeks, intermittent doses.

LITHIUM NON-CARCINOGENIC TOXICOLOGY

Overall Non-carcinogenic

Toxicology Rating: 1. Critical effect: Acute burn hazard (skin) from metallic lithium.

Lethal Dose: RTECS: LD₅₀ mouse: 1 g/kg, intraperitoneal.

Gosselin: "Acute oral LD₅₀ of lithium carbonate in rats is about 710 mg/kg, placing it near the borderline between [Gosselin] toxicity classes 3 and 4."

Toxic endpoints

associated with exposure: Gosselin: Central nervous system, kidneys, cardiovascular, muscles, poorly understood effects on the thyroid, known to produce nephrogenic diabetes insipidus, distal renal tubular acidosis, and impairment of renal concentrating ability.

Sax: Lithium ion has central nervous system toxicity.

NR CAPCOA, NR NIOSH, NR RTECS

Acute noncancer

reference exposure level: NR CAPCOA

NICKEL CARCINOGENICITY

Overall Carcinogenicity Rating: 1 (IARC 1 - nickel compounds as a group, not necessarily all compounds, EPA A - nickel refinery dust and nickel subsulfide); human carcinogen.

IARC Cancer Rating: IARC Supplement 7: IARC Group 1; human carcinogen (nickel compounds as a group, not necessarily all compounds).

EPA Cancer Weight-of-Evidence Group: IRIS: **Nickel refinery dust** rated Group A (Nickel refinery dust is a mixture of many nickel moieties, and it is not certain what the carcinogenic nickel species is in the refinery dust). Basis for classification: Human data in which exposure to nickel refinery dust caused lung and nasal tumors in sulfide nickel matte refinery workers in several epidemiologic studies in different countries, and on animal data in which carcinomas were produced in rats by inhalation and injection. (References²²).

IRIS: "Carcinogenicity assessment [for nickel] under review '94. The latest Health Assessment Document on Nickel states that the nickel ion (Ni+2) could be the ultimate carcinogenic form of nickel. Although this is unproven, it is considered prudent to make this assumption for covalent nickel (forms that generate Ni+2) and nickel salts. Proven carcinogenic forms of nickel are **nickel refinery dust** and **nickel subsulfide** (both in man) and nickel carbonyl (demonstrated in test animals). The former two substances are in weight-of-evidence group A, while the latter is in weight-of-evidence group B2. The salts of nickel show some carcinogenic activity. The testing of these nickel salts is inconclusive for assessment at this time due to limitations of the data base on these nickel salts, but since there is some cancer activity it is recommended by the HHAG that the hazard ranking under CERCLA be reported as "LOW". This "LOW" hazard ranking reflects the current data base on the nickel salts. -USEPA (1988) Methodology for Evaluating Potential Carcinogenicity in Support of Reportable Quantity Adjustments Pursuant to CERCLA Section 102, EPA/600/8-89/053."

Inhalation Cancer Potency Value: CAPCOA & OEHH: Inhalation unit risk: 2.6×10^{-4} per ($\mu\text{g}/\text{m}^3$) (nickel and nickel compounds). Calculated in document from potency slope of 9.1×10^{-1} ($\text{mg}/\text{kg}\cdot\text{day}$)⁻¹, by assuming a 70 kg person breathes 20 m³ of air per day.

HEAST: *Provisional* inhalation slope factor: 8.4×10^{-1} ($\text{mg}/\text{kg}\cdot\text{day}$)⁻¹ (for nickel refinery dust, based on respiratory system tumors in humans after occupational exposure).

IRIS: Inhalation unit risk: 2.4×10^{-4} per ($\mu\text{g}/\text{m}^3$).

Risk/Air Concentrations
Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
10^{-4} (1 in 10,000)	$4 \times 10^{-1} \mu\text{g}/\text{m}^3$
10^{-5} (1 in 100,000)	$4 \times 10^{-2} \mu\text{g}/\text{m}^3$
10^{-6} (1 in 1,000,000)	$4 \times 10^{-3} \mu\text{g}/\text{m}^3$

Non-Inhalation Cancer Potency Value: NR IRIS, NR OEHHA, NR CAPCOA

Carcinogenic Potential: Sax: "Confirmed carcinogen with experimental carcinogenic, neoplastigenic, tumorigenic, and teratogenic data."

RTECS: TD_{Lo} rat: 56 mg/kg, intramuscular. Tumorigenic (Carcinogenic by RTECS criteria; tumors at site of application, musculoskeletal tumors).
 TD_{Lo} rat: 250 mg/kg, implant. Tumorigenic (Carcinogenic by RTECS criteria; tumors at site of application, skin and appendages tumors).
 TD_{Lo} mouse: 200 mg/kg, intramuscular. Tumorigenic (Neoplastic by RTECS criteria; tumors at site of application).
 TD_{Lo} rabbit: 165 mg/kg/2 years-intermittent, implant. Tumorigenic (Neoplastic by RTECS criteria; uterine tumors, tumors at site of application).
 TD_{Lo} rat: 125 mg/kg/13 weeks-intermittent, intramuscular. Tumorigenic (Neoplastic by RTECS criteria; Tumors at site of application).
 TD_{Lo} rat: 800 mg/kg/13 weeks-intermittent, intramuscular. Tumorigenic (Neoplastic by RTECS criteria; musculoskeletal tumors, tumors at site of application).
 TD_{Lo} rat: 200 mg/kg/21 weeks-intermittent, intramuscular. Tumorigenic (Neoplastic by RTECS criteria; Tumors at site of application).
 TD_{Lo} rat: 1 g/kg/17 weeks-intermittent, intramuscular. Tumorigenic (Carcinogenic by RTECS criteria; Tumors at site of application).
(List truncated.)

IRIS: "HUMAN: Sufficient. **Nickel refinery dust** from pyrometallurgical sulfide nickel matte refineries is considered a human carcinogen when inhaled. Evidence of carcinogenicity includes a consistency of findings across different countries (Clydach, Wales; Copper Cliff, Ontario; Port Colborne, Ontario; Kristiansand, Norway; and Huntington, WV) in several epidemiologic studies, specificity of tumor site (lung and nose), high relative risks, particularly for nasal cancer, and a dose-response relationship by length of exposure. Excess risks are greatest in the dustier areas of the respective refineries. At Port Colborne, Roberts et al. (1983) reported high risks of lung (SMR = 298) and nasal (SMR = 9412) cancer among men ever exposed to calcining, leaching, and sintering, the dustier areas of the refinery. Similar exposures and high risks of lung and nasal cancer were observed in the calcining sheds at Clydach (lung SMR = 510, nasal SMR = 26,667) (Peto et al., 1984), the sintering furnaces at Copper Cliff (lung SMR = 424, nasal SMR = 1583) (Roberts and

Julian, 1982), and the roasting/smeltering (lung SMR = 360, nasal SMR = 4000) and electrolysis (lung SMR = 550, nasal SMR = 2700) furnaces at Kristiansand, Norway (Magnus et al., 1982). In the study of refinery and nonrefinery workers at a nickel refinery in West Virginia, nasal cancer was exclusive to the refinery workers, with an SMR of 2443 (Enterline and Marsh, 1982). No large excess of lung cancer was observed in either refinery (SMR = 118) or nonrefinery (SMR = 107.6) employees. The data do show a dose-response relationship between cumulative nickel exposure and lung cancer response (allowing for a 20-year latent period). The dose-response relationship is consistent with findings at nickel refineries in Clydach, Wales (Peto et al., 1984) and Copper Cliff, Ontario (Chovil et al., 1981). While the dust levels and lung cancer relative risks were much higher in the two latter refineries, all dose-response relationships appear linear, and the tumor type and sites are the same, indicating that the functional relationship spans a broad range of nickel exposures."

"ANIMAL: Animal studies indicate that some **nickel refinery dusts** are potentially carcinogenic. Nickel refinery flue dust (20% nickel sulfate, 59% nickel subsulfide, and 6.3% nickel oxide) from Port Colborne, Canada was tested for carcinogenic potential (Gilman and Ruckerbauer, 1962) by intramuscular injection. It was found to be a strong inducer of injection-site sarcomas in Hooded (52/66) and Wistar (8/20) rats after injection of 20 or 30 mg in one or both thighs and in mice (23/40) after injection of 10 mg/thigh. Fisher et al. (1971), as reviewed by Rigaut (1983), tested nickel refinery dust (20% nickel sulfate, 59% nickel subsulfide, and 6.3% nickel oxide) by inhalation. The refinery dust was one of six types of dust exposures administered to 348 rats at 5 to 15 mg/cu.m. The combined tumor incidence for refinery dust, synthetic dust, nickel subsulfide, and iron sulfide was 11 pulmonary tumors in the 348 rats. When Wistar rats were exposed to a combination of nickel and iron dust at concentrations of 2.1 ± 0.2 mg Ni/cu.m. and 1.9 ± 0.2 mg Fe/cu.m (Kim et al., 1976), one of the 60 surviving rats developed lung cancer."

"An intermediate of nickel refinery dust which contains nickel subsulfide, nickel oxide, and metallic nickel (Feinstein dust) was tested in albino (nonpedigree) rats at 70 mg dust/cu.m, 5 hours/day for 6 months (Saknyn and Blohkin, 1978, as reviewed by Sunderman, 1981). Squamous-cell carcinomas were found in two of the five surviving treated rats. Saknyn and Blohkin (1978) also treated the Albino rats by intraperitoneal injection of Feinstein dust at 90 to 150 mg/rat. Six of the 39 survivors developed injection-site sarcomas."

"Nickel dust from roasting (31% nickel subsulfide and 33.4% nickel oxide + silicon oxide and oxides of iron and aluminum) was tested for carcinogenicity in rats by inhalation (Belobragina and Saknyn, 1964, as reviewed by Rigaut, 1983). After exposure to 80 to 100 mg/cu.m, 5 hours/day for 12 months, no tumors were found."

"Three carcinogenicity studies (Schroeder and Mitchener, 1975; Schroeder et al., 1964, 1974) of nickel acetate and an unspecified nickel salt using doses of 5 ppm of nickel in the drinking water of Long-Evans rats and Swiss mice produced negative results. Ambrose et al. (1976) administered nickel sulfate hexahydrate in the diet of Wistar-derived rats and beagle dogs for 2 years at nickel concentrations of 100 to 2500 ppm. A lack of

carcinogenic response was observed in both studies. The dog study may have been inadequate to detect a carcinogenic response, since the duration was relatively short."

Mutagenic Potential: Sax: "Mutation data reported."

RTECS: Hamster/kidney: morphological transformation, 400 mg/L.
Hamster/embryo: morphological transformation, 5 μ mol/L.

NR IRIS

NICKEL NON-CARCINOGENIC TOXICOLOGY

Overall Non-carcinogenic

Toxicology Rating: 3. Critical effect: Immune system suppression. NOAEL 110 μ g/m³, inhalation, mouse.

Lethal Dose: Sax: LD₅₀ rat: 5 g/kg, oral.
LD₅₀ rat: 12 mg/kg, intratracheal.
LD₅₀ rat: 12.5 mg/kg, subcutaneous.
LD₅₀ mouse: 50 mg/kg, intravenous.
LD₅₀ dog: 10 mg/kg, intravenous.
LD₅₀ rabbit: 7 mg/kg, intraperitoneal.
LD₅₀ rabbit: 7.5 mg/kg, subcutaneous.
LD₅₀ guinea pig: 5 mg/kg, oral.

RTECS: Cited same studies as Sax, above, plus:
LD₅₀ cat: 12.5 mg/kg, subcutaneous. [Probably same as 12.5 mg/kg for rat, above.
Typographic error on database?]

Toxic endpoints

associated with exposure: CAPCOA: Nickel (all compounds) immune system, kidney, respiratory system.

Sax: Hypersensitivity common and can cause allergic contact dermatitis, pulmonary asthma, conjunctivitis. Poison by ingestion.

RTECS: Liver function tests impaired.

IRIS: Neonatal mortality, dermatotoxicity; kidney, liver, and spleen weights decreased in rats @ 35 mg/kg/day; While no reproductive effects have been associated with nickel exposure to humans, several studies in laboratory animals have demonstrated fetotoxicity.

Gosselin: Renal injury, central nervous system depression, skin, myocardial weakness (nickel salts).

NIOSH: Lungs, paranasal sinus, central nervous system (nickel metal and other compounds, as Ni).

Acute noncancer

reference exposure level: CAPCOA: Acute noncancer reference exposure level = $1.0 \times 10^{-6} \mu\text{g}/\text{m}^3$ for Nickel compounds. Toxic endpoint: immunotoxicity

Chronic noncancer

reference exposure level: CAPCOA: Inhalation: $2.4 \times 10^{-1} \mu\text{g}/\text{m}^3$ for Nickel and compounds (Derived from ACGIH TLV).

IRIS: Oral RfD = $2 \times 10^{-2} \text{ mg}/\text{kg}/\text{day}$, for nickel (soluble salts).

HEAST: *Provisional* subchronic oral RfD = $2 \times 10^{-2} \text{ mg}/\text{kg}/\text{day}$ (chronic oral RfD adopted as subchronic oral RfD).

1993-1994 ACGIH: Nickel, metallic, TLV-TWA = $1.0 \text{ mg}/\text{m}^3$.

**Critical effect upon which
acceptable exposure levels
are based/confidence level**

of critical study: IRIS: Critical effect for IRIS oral RfD for nickel (soluble salts) is decreased body and organ weight in exposed rats. NOAEL: 100 ppm diet (5 mg/kg/day). LOAEL: 1000 ppm diet (50 mg/kg/day).

Critical study in setting IRIS oral RfD for nickel (soluble salts): Ambrose, A.M., D.S. Larson, J.R. Borzelleca and G.R. Hennigar, Jr. (1976) Long-term Toxicologic Assessment of Nickel in Rats and Dogs. *J. Food Sci. Technol.* 13: 181-187.

"Oral RfD Confidence:
Study – Low
Data Base – Medium
RfD – Medium"

"An uncertainty factor of 10 is used for interspecies extrapolation and 10 to protect sensitive populations. An additional uncertainty factor of 3 is used to account for inadequacies in the reproductive studies. Total uncertainty factor used in calculating oral RfD: 300."

Teratogenicity: Sax: "An experimental teratogen"

TD_{Lo} rat: 158 mg/kg multi-generational study, oral. Teratogenic effects.

TD_{Lo} rat: 40 mg/kg/52 weeks, intermittent doses, parental. Teratogenic effects.

TD_{Lo} rabbit: 165 mg/kg/ 2 years, intermittent doses, implant. Teratogenic effects.

RTECS: TD_{Lo} rat: 158 mg/kg, oral, multigenerational study. Effects on fetus or embryo (fetotoxicity; fetal death) [also cited by Sax, above].

IRIS: While the systemic toxicity data (as manifested in organ weight changes) was used as the critical study for the RfD determination, the reproductive/fetotoxicity and the dermatotoxicity were both considered as possible endpoints upon which to base the quantitative risk assessment of nickel. The data for effects on the latter two endpoints do not demonstrate consistent dose-response relationships, and in both cases the available studies are sufficiently flawed so as to prevent their selection as the basis for the oral RfD.

Lewis: TD_{Lo} rat: 158 mg/kg, oral. Fetotoxicity (stunted fetus).
Teratogenic effects listed for certain nickel compounds.

Hepatotoxicity: RTECS: Multiple dose toxicity study: TC_{Lo} rat: 100 µg/m³/24 hours /17 weeks constant, liver function tests impaired.

IRIS: Dietary studies show reduction in liver weights in exposed rats. EPA is proposing to regulate nickel based on this potential adverse effect.

NR Sax, NR Zimmerman, NR Meeks

Immunotoxicity: Nickel sulfate: Can cause asthma in humans. On European Environmental and Contact Dermatitis Research Group's (EECDRG), and International Contact Dermatitis Research Group (ICDRG) recommended test list.²³

Nickel (inorganic; sulfide and chloride): Reduces lymphatic organ weights, decreases formation of interferon, decreases cell-mediated immunity and lymphocyte blast formation.²⁴

CAPCOA: Nickel (all compounds) immune system as target organ.

Sax: Hypersensitivity common and can cause allergic contact dermatitis

Gosselin Toxicity Rating: Metallic or elemental nickel not rated. **Nickel salts** (i.e. nickel chloride, nickel phosphate, nickel sulphate) rated **4 (?)**. **Nickel carbonyl** (also a carcinogen) rated **6**.

SODIUM CARCINOGENICITY

Overall Carcinogenicity Rating: 3; not classifiable.

IARC Cancer Rating: NR IARC

EPA Cancer Weight-of-Evidence Group: NR EPA, NR IRIS

Inhalation Cancer Potency Value: NR IRIS, NR CAPCOA, NR OEHHA

Non-Inhalation Cancer Potency Value: NR IRIS, NR CAPCOA, NR OEHHA

Carcinogenic Potential: NR RTECS, NR Sax, NR IRIS

Mutagenic Potential: NR RTECS, NR Sax, NR IRIS

SODIUM NON-CARCINOGENIC TOXICOLOGY

Overall Non-carcinogenic

Toxicology Rating: 1. Critical effect: Acute burn hazard (skin) from metallic sodium.

Lethal Dose: NR Sax

RTECS: LD₅₀ mouse: 4 g/kg, intraperitoneal.

Toxic endpoints

associated with exposure: NR CAPCOA, NR NIOSH, NR RTECS

Sax: "Metallic sodium reacts exothermically with moisture of the body or tissue surfaces, causing thermal and chemical burns."

Acute noncancer

reference exposure level: NR CAPCOA

Chronic noncancer

reference exposure level: NR CAPCOA, NR IRIS, NR ACGIH TLV-TWA

**Critical effect upon which
acceptable exposure levels
are based/confidence level
of critical study:** NR

Teratogenicity: NR RTECS, NR IRIS, NR Sax

Lewis: NR as sodium. Teratogenicity noted for various sodium compounds.

Hepatotoxicity: NR RTECS, NR IRIS, NR Sax, NR Zimmerman, NR Meeks

Immunotoxicity: NR IRIS

Gosselin Toxicity Rating: Elemental or metallic sodium not rated. Toxicity of sodium compounds varies.

Miscellaneous: Keep away from water at all times. Dangerous explosion hazard when metallic sodium exposed to moisture in any form. Elemental sodium is highly reactive.

SULFUR CARCINOGENICITY

Overall Carcinogenicity Rating: 3; not classifiable.

IARC Cancer Rating: NR IARC

EPA Cancer Weight-of-Evidence Group: NR EPA, NR IRIS

Inhalation Cancer Potency Value: NR OEHHA, NR CAPCOA, NR IRIS

Non-Inhalation Cancer Potency Value: NR OEHHA, NR CAPCOA, NR IRIS

Carcinogenic Potential: NR RTECS, NR IRIS, NR Sax

Mutagenic Potential: NR RTECS, NR IRIS, NR Sax

SULFUR NON-CARCINOGENIC TOXICOLOGY

Overall Non-carcinogenic

Toxicology Rating: 5. Critical effect: Eye irritation (skin) @ 10,640 $\mu\text{g}/\text{m}^3$, human.

Lethal Dose: Sax: LD_{Lo} rat: 8 mg/kg, intravenous.

LD_{Lo} dog: 10 mg/kg, intravenous.

LD_{Lo} rabbit: 5 mg/kg, intravenous.

LD_{Lo} rabbit: 175 mg/kg, oral.

LD_{Lo} guinea pig: 55 mg/kg, intraperitoneal.

RTECS: Cited same studies as Sax, above, plus:

LD rat: >8437 mg/kg, oral.

Toxic endpoints

associated with exposure: Sax: Poison by ingestion, intravenous, or intraperitoneal routes. Human eye and mucous membrane irritant. Human eye effects at 8 ppm (10.64 mg/m^3).

RTECS: Human eye irritant.

Gosselin: Irritation to skin, eye, and respiratory tract. "Large doses orally may lead to production of hydrogen sulfide *in vivo*, due to bacterial action in the colon."

NR CAPCOA, NR NIOSH

Acute noncancer

reference exposure level: NR CAPCOA

Chronic noncancer

reference exposure level: NR CAPCOA, NR IRIS, NR ACGIH TLV-TWA

**Critical effect upon which
acceptable exposure levels
are based/confidence level
of critical study:** NR

Teratogenicity: NR RTECS, NR Sax, NR IRIS, NR Lewis

Hepatotoxicity: Meeks: "The liver is subject to direct injurious attack by a wide variety of so-called primary hepatotoxins, including...sulfur compounds."

NR RTECS, NR Sax, NR IRIS, NR Zimmerman

Immunotoxicity: NR IRIS

Gosselin Toxicity Rating: 3(?). Low toxicity, but may cause irritation to skin, eye, and respiratory tract. Large doses (15 g) orally may lead to hydrogen sulfide production *in vivo*, specifically in the colon. Small particles are more toxic than large ones, orally. A man has survived the ingestion of 60 g of sulfur over a period of 24 hours.

SULFURIC ACID CARCINOGENICITY

Overall Carcinogenicity Rating: 1 (IARC 1, NR EPA); human carcinogen.

IARC Cancer Rating: IARC Group 1; human carcinogen (strong inorganic acid mists containing sulfuric acid).²⁵

EPA Cancer Weight-of-Evidence Group: NR EPA, NR IRIS

Inhalation Cancer Potency Value: NR OEHHA, NR IRIS, NR CAPCOA

Non-Inhalation Cancer Potency Value: NR OEHHA, NR IRIS, NR CAPCOA

Carcinogenic Potential: NR RTECS, NR IRIS, NR Sax

Mutagenic Potential: RTECS: Hamster/ovary: cytogenic analysis, 4 mmol/L.

NR IRIS, NR Sax

SULFURIC ACID NON-CARCINOGENIC TOXICOLOGY

Overall Non-carcinogenic

Toxicology Rating: 1. Critical effect: Acute burn hazard (skin).

Lethal Dose: Sax: Sulfuric Acid:

LD_{Lo} man: 135 mg/kg, route of administration unreported.

LD₅₀ rat: 2140 mg/kg, oral.

LC₅₀ rat: 510 mg/m³/2 hours, inhalation.

LC₅₀ mouse: 320 mg/m³/2 hours, inhalation.

LC₅₀ guinea pig: 18 mg/m³, inhalation.

RTECS: Cited same studies as Sax, above.

Gosselin: "A reasonable estimate of the lethal dose in an adult is 1 oz. sulfuric acid in a 95% solution. However, a few milliliters [of mineral acid, in general] has killed, and even a few drops presents a hazard if aspirated into the larynx."

Sax: Sulfuric Acid (fuming):

LC₅₀ rat: 347 ppm/1 hour, inhalation.

Toxic endpoints

associated with exposure: Gosselin: "Corrosive burns may result from the inhalation of acid fumes and from skin contact with or the ingestion of strong acids."

Sax: "Repeated or prolonged inhalation of a mist of sulfuric acid can cause inflammation of the upper respiratory tract leading to chronic bronchitis...Severe exposure may cause chemical pneumonitis...erosion of teeth due to exposure to strong acid fumes."

NIOSH: Respiratory system, eyes, skin, teeth.

RTECS: Lungs, changes in teeth and supporting structures.

NR CAPCOA

Acute noncancer

reference exposure level: NR CAPCOA

Chronic noncancer

reference exposure level: HEAST: *Provisional* chronic reference concentration comment: "Reported effects [respiratory effects] occurred at portal of entry [human]. Estimates of mg/day RfDs are inappropriate because effects at portal of entry depend on concentration in air. An acceptable air concentration of 0.07 mg/m³ was estimated by Carson, *et al.* (1981) from available data."

1993-1994 ACGIH: TLV-TWA = 1.0 mg/m³.

NR CAPCOA, NR IRIS

**Critical effect upon which
acceptable exposure levels
are based/confidence level**

of critical study: HEAST: *Provisional* chronic reference concentration based on respiratory effects at portal of entry.

1991 ACGIH: TLV-TWA of 1.0 mg/m³ based on potential for pulmonary irritation.

NR IRIS

Teratogenicity: RTECS: TC_{LD} rabbit: 20 mg/m³/7 hours (6-18 days preg.), inhalation. Specific developmental abnormalities (musculoskeletal system).

Lewis: Cites same study as RTECS, above.

Sax: "An experimental teratogen." Cites same study as RTECS, above.

NR IRIS

Hepatotoxicity: NR RTECS, NR Sax, NR IRIS, NR Zimmerman, NR Meeks

Immunotoxicity: NR IRIS

Gosselin Toxicity Rating: Not rated. Corrosive agents are not usually rated by Gosselin Toxicity Rating, since death is usually the result of severe local tissue injury and associated complications.

VANADIUM CARCINOGENICITY

Overall Carcinogenicity Rating: 3; not classifiable.

IARC Cancer Rating: NR IARC

EPA Cancer Weight-of-Evidence Group: NR EPA, NR IRIS

Inhalation Cancer Potency Value: NR IRIS, NR CAPCOA, NR OEHHA

Non-Inhalation Cancer Potency Value: NR IRIS, NR CAPCOA, NR OEHHA

Carcinogenic Potential: RTECS: No unequivocal studies.

Sax: No unequivocal studies. "Questionable carcinogen with experimental tumorigenic data."

NR IRIS

Mutagenic Potential: NR RTECS, NR IRIS

Sax: "Some [vanadium] compounds have reported mutagenic effects."

VANADIUM NON-CARCINOGENIC TOXICOLOGY

Overall Non-carcinogenic

Toxicology Rating: 3. Critical effect: Secondary bronchitis (respiratory) @ $>50 \mu\text{g}/\text{m}^3$ /chronic occupational exposure (most common symptom), inhalation, human. Based on vanadium pentoxide.

Lethal Dose: Sax: LD_{50} rabbit: 59 mg/kg, subcutaneous.

RTECS: Cited same study as Sax, above.

1991 ACGIH: "The pentavalent compounds, such as vanadium pentoxide and vanadates, are more toxic than other forms." LD rabbit: 1.5 mg/kg, intravenous vanadium pentoxide. LD_{50} mouse: 23 mg/kg, oral vanadium pentoxide.

Toxic endpoints

associated with exposure: NR CAPCOA, NR RTECS

Sax: "Vanadium compounds act chiefly as an irritant to the conjunctivae and respiratory tract... There is still some controversy as to the effects of industrial exposure on other systems of the body."

NIOSH: Respiratory system, skin, eyes (vanadium pentoxide, respirable dust or fume as V_2O_5 - vanadium pentoxide).

Acute noncancer

reference exposure level: NR CAPCOA

Chronic noncancer

reference exposure level: IRIS: Oral RfD: A risk assessment for this substance/agent is under review by an EPA work group [1994].

HEAST: *Provisional* chronic and subchronic oral RfDs are both 7×10^{-3} mg/kg/day.

1993-1994 ACGIH: Vanadium pentoxide, as V_2O_5 (respirable dust or fume), TLV-TWA = 0.05 mg/m^3 .

NR CAPCOA

**Critical effect upon which
acceptable exposure levels
are based/confidence level**

of critical study: IRIS: See above.

HEAST: *Provisional* chronic and subchronic oral RfDs of 7×10^{-3} mg/kg/day are based upon oral drinking water (lifetime) study of rat; NOAEL = 5 ppm. Uncertainty factor of 100 applied.

1991 ACGIH: TLV-TWA of 0.05 mg/m^3 for vanadium pentoxide based on potential of upper respiratory tract irritation.

Teratogenicity: NR IRIS, NR RTECS, NR Sax

Lewis: Vanadium pentoxide (dust). TD_{Lo} mouse: 10900 mg/kg (8 days preg.), intravenous. Specific developmental abnormalities (musculoskeletal).

Hepatotoxicity: NR RTECS, NR IRIS, NR Sax, NR Zimmerman, NR Meeks

Immunotoxicity: Vanadium trioxide and vanadium pentoxide can cause asthma in humans.

Gosselin Toxicity Rating: "5. Has a toxicity of about the same magnitude as pentavalent arsenic. Poisoning from dust inhalation is fairly common in industry. Anionic vanadium is said to be more toxic than cationic. Humans have tolerated doses of 150 mg of vanadium sodium tartrate intramuscularly, and 1 to 8 mg of sodium metavanadate orally."

ZINC CARCINOGENICITY

Overall Carcinogenicity Rating: 3 (EPA D); not classifiable.

IARC Cancer Rating: NR IARC

EPA Cancer Weight-of-Evidence Group: IRIS: D; not classifiable as to human carcinogenicity. Basis for classification: "Based on inadequate evidence in humans and animals. There are no reports on the possible carcinogenicity of zinc and compounds per se in humans. Case studies have been used to evaluate the effects of zinc administered for therapeutic reasons. There are reports which compare zinc levels in normal and cancerous tissue. Studies of occupational exposure to zinc compounds have also been conducted, but have limited value because they do not correlate exposure with cancer risk." (References²⁶).

Inhalation Cancer Potency Value: NR CAPCOA, NR IRIS, NR OEHHA

Non-Inhalation Cancer Potency Value: NR CAPCOA, NR IRIS, NR OEHHA

Carcinogenic Potential: IRIS: "No statistically significant reports of elevated tumorigenicity."

NR RTECS

Mutagenic Potential:

IRIS: "The results of short-term genotoxicity assays for zinc are equivocal."

"Zinc chloride is reported to be positive in the Salmonella assay (Kalinina, *et al.*, 1977), negative in the mouse lymphoma assay (Amacher and Paillet, 1980), and a weak clastogen[†] in cultured human lymphocytes (Deknudt and Deminatti, 1978). Zinc sulfate is reported to be not mutagenic in the Salmonella assay (Gocke, *et al.*, 1981), and zinc acetate is reported to not induce chromosomal aberrations in cultured human lymphocytes (Gasiorek and Bauchinger, 1981). Crebelli, *et al.* (1985) found zinc oxide (99% purity) (1-5 mg/plate) to be not mutagenic for Salmonella in the reversion assay."

"Responses in mutagenicity assays are thought to depend on the form (e.g., inorganic or organic salt) of the zinc tested. For example, inorganic salts tend to dissociate and the zinc becomes bound with culture media constituents. Salts that dissociate less readily tend to be transported into the cell and are postulated to cause a positive response."

RTECS: EPA Genetox Program 1988: inconclusive, *in vivo* cytogenics-human lymphocyte.

[†] Clastogenic compounds induce chromosome breakages.

ZINC NON-CARCINOGENIC TOXICOLOGY

Overall Non-carcinogenic

Toxicology Rating: 3. Critical effect: Respiratory irritation @ 350 $\mu\text{g}/\text{m}^3$, inhalation, human. Based on zinc compounds.

Lethal Dose: RTECS: No lethal dose reported. TC_{50} human inhalation: 124 $\text{mg}/\text{m}^3/50$ minutes, causes cough, dyspnea, sweating.

Sax: "Zinc is not inherently a toxic element. Zinc Compounds have variable, usually low, toxicity."

Toxic endpoints

associated with exposure: CAPCOA: Cardiovascular or blood system, respiratory system.

Sax: Skin irritant, human systemic effects by ingestion (cough, dyspnea, sweating).

RTECS: Skin irritant, respiratory irritant.

NIOSH: Respiratory system (zinc oxide fume).

Acute noncancer

reference exposure level: NR CAPCOA

Chronic noncancer

reference exposure level: CAPCOA: $3.5 \times 10^{-1} \mu\text{g}/\text{m}^3$ (inhalation) for Zinc compounds.

**Critical effect upon which
acceptable exposure levels
are based/confidence level
of critical study:** NR CAPCOA

Teratogenicity: NR RTECS, NR IRIS, NR Sax

Lewis: NR as zinc. Teratogenic effects noted for certain zinc compounds.

Hepatotoxicity: NR RTECS, NR IRIS, NR Sax, NR Zimmerman, NR Meeks

Immunotoxicity: Zinc is an immunomodulator²⁷. Stimulation at low dose followed by a depression at higher doses. At high doses, decreased antibody production and blastogenesis^u of splenic cells were reported.²⁸

^u Blastogenesis is the morphological transformation of small lymphocytes into large cells resembling blast cells, occurring on exposure to antigens to which the donor is immunized.

Gosselin Toxicity Rating: Elemental zinc not rated. Gosselin Toxicity Rating for **soluble zinc salts** (i.e., chloride, sulfate, acetate): **3 to 4**. Produces irritation or corrosion of the alimentary tract with pain, emesis, etc. The chloride appears to be more corrosive and more toxic than the sulfate. A few grams of the chloride have killed an adult, although recovery has been reported after the ingestion of 90 grams.

ENDNOTES:

1. References for EPA carcinogenicity assessment of cadmium listed in IRIS, Online, 12/94:

Armstrong, B.G. and G. Kazantzis. 1983. The mortality of cadmium workers. *Lancet*. June 25, 1983: 1425-1427.

Casto, B. 1976. Letter to Richard Troast, U.S. EPA. Enclosing mutagenicity data on cadmium chloride and cadmium acetate.

Gilliavod, N. and A. Leonard. 1975. Mutagenicity tests with cadmium in the mouse. *Toxicology*. 5: 43-47.

Holden, H. 1980. Further mortality studies on workers exposed to cadmium fumes. Presented at Seminar on Occupational Exposure to Cadmium, March 20, 1980, London, England.

Kipling, M.D. and J.A.H. Waterhouse. 1967. Cadmium and prostatic carcinoma. *Lancet*. 1: 730.

Lemen, R.A., J.S. Lee, J.K. Wagoner and H.P. Blejer. 1976. Cancer mortality among cadmium production workers. *Ann. N.Y. Acad. Sci.* 271: 273.

Oberly, T., C.E. Piper and D.S. McDonald. 1982. Mutagenicity of metal salts in the L5178 Y mouse lymphoma assay. *J. Toxicol. Environ. Health*. 9: 367-376.

Ochi, T. and M. Ohsawa. 1983. Induction of 6-thioguanine-resistant mutants and single-strand scission DNA by cadmium chloride in cultured Chinese hamster cells. *Mutat. Res.* 111: 69-78.

Sanders, C.L. and J.A. Mahaffey. 1984. Carcinogenicity of single and multiple intratracheal instillations of cadmium oxide in the rat. *Environ. Res.* 33: 227-233.

Shimada, T., T. Watanabe and A. Endo. 1976. Potential mutagenicity of cadmium in mammalian oocytes. *Mutat. Res.* 40: 389-396.

Sorahan, T. and J.A.H. Waterhouse. 1983. Mortality study of nickel-cadmium battery workers by the method of regression models in life tables. *Br. J. Ind. Med.* 40: 293-300.

Takenaka, S., H. Oldiges, H. Konig, D. Hochrainer and G. Oberdoerster. 1983. Carcinogenicity of cadmium aerosols in Wistar rats. *J. Natl. Cancer Inst.* 70: 367-373.

Thun, M.J., T.M. Schnorr, A.B. Smith and W.E. Halperin. 1985. Mortality among a cohort of U.S. cadmium production workers: An update. *J. Natl. Cancer Inst.* 74(2): 325-333.

U.S. EPA. 1985. Updated Mutagenicity and Carcinogenicity Assessment of Cadmium. Addendum to the Health Assessment Document for Cadmium (EPA 600/B- B1-023). EPA 600/B-83-025F.

Varner, M.O. 1983. Updated epidemiologic study of cadmium smelter workers. Presented at the Fourth International Cadmium Conference. Unpublished.

Watanabe, T., T. Shimada and A. Endo. 1979. Mutagenic effects of cadmium on mammalian oocyte chromosomes. *Mutat. Res.* 67: 349-356.

2. Lewis, R.M. and Cavagnaro, J. (1991) The Implications of Age-Related Changes in Immune Function and Toxicity, *Aging and Environmental Toxicology*, Johns Hopkins University Press, Baltimore, pp. 107-124.

3. Cifone, M.G., et al. (1990) Cadmium Inhibits Spontaneous (NK), Antibody-mediated (ADCC) and IL-2-stimulated Cytotoxic Functions of Natural Killer Cells, *Immunopharmacology* 20:73-80 [abstract].

4. Chowdhury, B.A. and Chandra, R.K. (1989) Effect of Zinc Administration on Cadmium-induced Suppression of Natural Killer Cell Activity in Mice, *Immunol. Letters* 22:287-91 [abstract].

5. United States Environmental Protection Agency (1994) *Health Assessment Document for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds, Review Draft.*

6. Birnbaum, Linda S. (1994) The Mechanism of Dioxin Toxicity: Relationship to Risk Assessment, *Environmental Health Perspectives* 102:157-167.

7. RTECS On-line, 1990.

8. Lewis, R.M. and Cavagnaro, J. (1991) The Implications of Age-Related Changes in Immune Function and Toxicity, *Aging and Environmental Toxicology*, Johns Hopkins University Press, Baltimore, pp. 107-124.

9. References for EPA carcinogenicity assessment of lead listed in IRIS, Online, 12/94:

Anderson, E.L., and CAG (Carcinogenic Assessment Group). 1983. Quantitative approaches in use to assess cancer risk. *Risk Analysis*. 3: 277-295.

Azar, A., H.J. Trochimowicz and M.E. Maxfield. 1973. Review of lead studies in animals carried out at Haskell Laboratory - Two year feeding study and response to hemorrhage study. In: Barth D., A. Berlin, R. Engel, P. Recht and J. Smeets, Ed. *Environmental health aspects of lead: Proceedings International Symposium; October 1972; Amsterdam, The Netherlands. Commission of the European Communities, Luxemburg.* p. 199-208.

Casto, B.C., J. Meyers and J.A. DiPaolo. 1979. Enhancement of viral transformation for

evaluation of the carcinogenic or mutagenic potential of inorganic metal salts. *Cancer Res.* 39: 193-198.

Cooper, W.C. 1985. Mortality among employees of lead battery plants and lead producing plants, 1947-1980. *Scand. J. Work Environ. Health.* 11: 331-345.

Cooper, W.C. and W.R. Gaffey. 1975. Mortality of lead workers. In: *Proceedings of the 1974 Conference on Standards of Occupational Lead Exposure*, J.F. Cole, Ed., February, 1974. Washington, DC. *J. Occup. Med.* 17: 100-107.

Dingwall-Fordyce, I. and R.E. Lane. 1963. A follow-up study of lead workers. *Br. J. Ind. Med.* 20: 313-315.

DiPaolo, J.A., R.L. Nelson and B.C. Casto. 1978. In vitro neoplastic transformation of Syrian hamster cells by lead acetate and its relevance to environmental carcinogenesis. *Br. J. Cancer.* 38: 452-455.

Grandjean, P., H.C. Wulf and E. Niebuhr. 1983. Sister chromatid exchange in response to variations in occupational lead exposure. *Environ. Res.* 32: 199-204.

Kasprzak, K.S., K.L. Hoover and L.A. Poirier. 1985. Effects of dietary calcium acetate on lead subacetate carcinogenicity in kidneys of male Sprague-Dawley rats. *Carcinogenesis.* 6(2): 279-282.

Koller, L.D., N.I. Kerkvliet and J.H. Exon. 1986. Neoplasia induced in male rats fed lead acetate, ethyl urea and sodium nitrate. *Toxicol. Pathol.* 13: 50-57.

Nelson, D.J., L. Kiremidjian-Schumacher and G. Stotzky. 1982. Effects of cadmium, lead, and zinc on macrophage-mediated cytotoxicity toward tumor cells. *Environ. Res.* 28: 154-163.

Selevan, S.G., P.J. Landrigan, F.B. Stern and J.H. Jones. 1985. Mortality of lead smelter workers. *Am. J. Epidemiol.* 122: 673-683.

U.S. EPA. 1984. *Health Effects Assessment for Lead*. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC. EPA/540/1-86/055. NTIS PB85-163996/AS.

U.S. EPA. 1986. *Air Quality Criteria Document for Lead. Volumes III, IV*. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC, for the Office of Air Quality Planning and Standards. EPA-600/8-83/028dF.

U.S. EPA. 1989. *Evaluation of the potential Carcinogenicity of lead and lead compounds: In support of reportable quantity adjustments pursuant to CERCLA Section 102*. Prepared

by the Office of Health and Environmental Assessment, Washington, DC. EPA/600/8-89/045A. (External Review Draft).

Van Esch, G.J. and R. Kroes. 1969. The induction of renal tumors by feeding of basic lead acetate to mice and hamsters. *Br. J. Cancer*. 23: 265-271.

10. Lewis, R.M. and Cavagnaro, J. (1991) The Implications of Age-Related Changes in Immune Function and Toxicity, *Aging and Environmental Toxicology*, Johns Hopkins University Press, Baltimore, pp. 107-124.

11. Queiroz, M.L., et al. (Defective Neutrophil Function in Workers Occupationally exposed to Lead, *Pharmacology and Toxicology* 72:73-77 [abstract].

12. Fischbein, A., et al. (1993) Phenotypic Aberrations of CD3+ and CD4+ Cells and Functional Impairments of Lymphocytes at Low-Level Occupational Exposure to Lead, *Clinical Immunology and Immunopathology* 66:163-168 [abstract].

13. Governa, M., et al. (1987) In Vitro Impairment of Human Granulocyte Functions by Lead, *Archives of Toxicology* 59:421-425 [abstract].

14. Governa, M., et al. (1988) Impairment of Chemotaxis of Polymorphonuclear Leukocytes from Lead Acid Battery Workers, *Science of the Total Environment* 71:543-546 [abstract].

15. Kowolenko, M., et al. (1988) Effect of Lead on Macrophage Function, *Journal Leukoc. Biology* 43:357-364 [abstract].

16. McCabe, M.J., Jr., & Lawrence, D.J. (1990) The Heavy Metal Lead Exhibits B-Cell-stimulatory Factor Activity by Enhancing B-Cell in Expression and Differentiation, *Journal of Immunology* 45:671-677 [abstract].

17. Bull. Inst. Marit. Trop. Med. Gdynia 41:27-36 [abstract].

18. Koller, Loren (1990) The Immunotoxic Effects of Lead in Lead-Exposed Laboratory Animals, *Annals of the New York Academy of Sciences* 587:160-167.

19. Faith, R.E., et al. Effect of Chronic Developmental Lead Exposure on Cell-Mediated Immune Functions, *Clin. Exp. Immunol.* 35:413-420.

20. Kowolenko, M., et al. (1988) Effect of Lead on Macrophage Function, *Journal Leuc. Biol.* 43:357-364.

21. Ellenhorn, Matthew J. & Barceloux, Donald G. (1988) *Medical Toxicology: Diagnosis and Treatment of Human Poisoning*, Elsevier, New York.

22. References for EPA carcinogenicity assessment of nickel refinery dust listed in IRIS, Online, 12/94:

Ambrose, A.M., P.S. Larson, J.F. Borzelleca and G.R. Hennigar. 1976. Long term toxicologic assessment of nickel in rats and dogs. *J. Food Sci. Tech.* 13: 181-187.

Belobragina, G.V. and A.V. Saknyn. 1964. An experimental study into the effects produced by dust in nickel industry. *Gig. Tr. Prof. Zabol.* 7:8 (1963), *Abst. Bull. Hyg.* 39: 497.

Chovil, A., R.B. Sutherland and M. Halliday. 1981. Respiratory cancer in a cohort of sinter plant workers. *Br. J. Ind. Med.* 38: 327-333.

Enterline, P.E. and G.M. Marsh. 1982. Mortality among workers in a nickel refinery and alloy manufacturing plant in West Virginia. *J. Natl. Cancer Inst.* 68(6): 925-933.

Gilman, J.P.W. and G.M. Ruckerbauer. 1962. Metal carcinogenesis. I. Observations on the carcinogenicity of a refinery dust, cobalt oxide, and colloidal thorium dioxide. *Cancer Res.* 22: 152-157.

Kim, M.K., A.M. Fisher and R.J. Mackay. 1976. Pulmonary effects of metallic dusts--nickel and iron [master's thesis]. University of Toronto, Toronto, Ontario, Canada.

Magnus, K., A. Andersen and A. Hogetveit. 1982. Cancer of respiratory organs among workers at a nickel refinery in Norway. *Int. J. Cancer.* 30: 681-685.

Peto, J., H. Cuckle, R. Doll, C. Hermon and L.G. Morgan. 1984. Respiratory cancer mortality of Welsh nickel refinery workers. In: *Nickel in the Human Environment: Proceedings of a Joint Symposium, March, 1983. IARC Scientific Publ. No. 53. International Agency for Research on Cancer, Lyon, France, p. 36-46.*

Rigaut, J.P. 1983. Rapport preparatoire sur Les criteres de Sante Pour le nickel. Doc. CCE/Lux/V/E/24/83. (Trans. Fre.)

Roberts, R.S. and J.A. Julian. 1982. Mortality study of Canadian nickel miners. In: *Proceedings of the Conference on Health Issues-related to Metal and Non-metallic Mining. Wagner, W.L., W.N. Rom and J.A. Merchant Eds. Park City, UT. p.241-260.*

Roberts, R.S., J.A. Julian, D.C.F. Muir and H. Shannon. 1983. Cancer mortality associated with nickel sintering. Occupational Health Faculty of Health Sciences. McMaster University Hamilton, Ontario, Canada. Presented at the IARC Nickel Symposium March 1983, Lyon, France.

Sakun, A.V. and V.A. Blohkin. 1978. On the development of malignant tumors in rats exposed to nickel containing aerosols. *Vopr. Onkol.* 24(4): 44-48.

Schroeder, H.A., J.J. Balassa and W.H. Vinton Jr. 1964. Chromium, lead, cadmium, nickel and titanium in mice: Effect on mortality, tumors and tissue levels. *J. Nutr.* 83: 239-250.

Schroeder, H.A., M. Mitchener and A.P. Nason. 1974. Life-term effects of nickel in rats:

Survival, tumors, interactions with trace elements and tissue levels. *J. Nutr.* 104: 239-243.

Schroeder, H.A. and M. Mitchener. 1975. Life-term effects of mercury, methyl mercury, and nine other trace metals on mice. *J. Nutr.* 105: 452-458.

Sunderman, F.W. 1981. Recent research on nickel carcinogenesis. *Environ. Health Perspect.* 40: 131-141.

U.S. EPA. 1986. Health Assessment Document for Nickel and Nickel Compounds. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA/600/8- 83/012FF.

23. Anonymous (1988) Notice: Revised European Standard Series from 1 January 1989, *Contact Dermatitis* 19:391.

24. Sonnenfeld, G., et al. (1983) Differential Effects of Amorphous and Crystalline Nickel Sulfide on Murine Interferon Production, *Environmental Research* 32:474-479.

Smialowicz, R.J., et al. (1984) Immunologic Effects of Nickel: 1. Suppression of Cellular and Humoral Immunity, *Environ. Res.* 33:413-427.

McConnell, L.H., et al. (1973) Asthma Caused by Nickel Sensitivity, *Ann. Intern. Med.* 78:888-890.

25. IARC, World Health Organization, (1989) *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*, Volume 54, Lyon, France.

26. References for EPA carcinogenicity assessment of zinc listed in IRIS, Online, 12/94:

Amacher, D.E. and S.C. Paillet. 1980. Induction of trifluorothymidine-resistant mutants by metal ions in L5178Y/TK+/- cells. *Mutat. Res.* 78: 279-288.

Aughey, E., L. Grant, B.L. Furman and W.F. Dryden. 1977. The effects of oral zinc supplementation in the mouse. *J. Comp. Pathol.* 87:1-14.

Batchelor, R.P., J.W. Fehnel, R.M. Thomson and K.R. Drinker. 1926. A clinical and laboratory investigation of the effect of metallic zinc, of zinc oxide and of zinc sulphide upon the health of workmen. *J. Ind. Hyg.* 8: 322-363. (Summarized in U.S. EPA, 1987)

Bobrishchev-Pushkin, D.M., A.A. Orlova, L.A. Naumova et al. 1977. Occupational hygiene and health status of workers engaged in production of copper alloys. *Gig. Tr. Prof. Zabol.* (8): 10-14. (Eng. trans.)

Chmielewski, J., B. Jaremin, C. Bartnicki and R. Konieczka. 1974a. Evaluation of occupational exposure to zinc oxide in the marine production shipyard: I. Examination of the working environment and the stands under exposure. *Biul. Inst. Med. Morsk. Gdansk.* 25(1): 43-51. (Summarized in U.S. EPA, 1987)

Chmielewski, J., B. Jaremin, C. Bartnicki and R. Konieczka. 1974b. Evaluation of occupational exposure to zinc oxide in the marine production shipyard: II. Examination of the state of health of the workers exposed to zinc oxide. *Biul. Inst. Med. Morsk. Gdansk.* 25(1): 53-65. (Summarized in U.S. EPA, 1987)

Crebelli, R., A. Paoletti, E. Falcone, G. Aquilina, G. Fabri and A. Carere. 1985. Mutagenicity studies in a tyre plant: In vitro activity of workers' urinary concentrates and raw materials. *Br. J. Ind. Med.* 42: 481-487.

Deknuddt, G. and M. Deminatti. 1978. Chromosome studies in human lymphocytes after in vitro exposure to metal salts. *Toxicology.* 10: 67-75.

Fong, L.Y.Y., A. Sivak and P.M. Newberne. 1978. Zinc deficiency and methylbenzyl-nitrosoamine-induced esophageal cancer in rats. *J. Natl. Cancer Inst.* 61: 145-150.

Gasiorek, K. and M. Bauchinger. 1981. Chromosome changes in human lymphocytes after separate and combined treatment with divalent salts of lead, cadmium, and zinc. *Environ. Mutagen.* 3: 513-518.

Gocke, E., M.T. King, K. Echardt and D. Wild. 1981. Mutagenicity of cosmetics ingredients licensed by the European Communities. *Mutat. Res.* 90: 91-109.

Guthrie, J. 1956. Attempts to produce seminomata in the albino rat by inoculation of hydrocarbons and other carcinogens into normally situated and ectopic testes. *Br. J. Cancer.* 10: 134-144.

Guthrie, J. 1964. Observations on the zinc induced testicular teratomas of fowl. *Br. J. Cancer.* 18: 130-142.

Habib, F.K., G.L. Hammond, I.R. Lee et al. 1976. Metal-androgen interrelationships in carcinoma and hyperplasia of the human prostate. *J. Endocr.* 71: 133-141.

Halme, E. 1961. On the carcinogenic effect of drinking water containing zinc. *Vitalstoffe.* 6: 59-66. (Ger.) (Eng. trans.)

Kalinina, L.M., G.H. Polukhina and L.I. Lukasheva. 1977. Salmonella Typhimurium - Test system for indication of mutagenic activity of environmental hazards. I. Detection of mutagenic effect of heavy metal salts using in vivo and in vitro assays without metabolic activation. *Genetika.* 13: 1089-1092.

Klucik, I. and J. Koprda. 1979. Hypocalcaemia in subjects after long-term exposure to zinc oxide. *Prac. Lek.* 31(6): 234-237. (Summarized in U.S. EPA, 1987)

Mathur, A., K. Wallenius, and M. Abdulla. 1979. Influence of zinc on onset and progression of oral carcinogenesis in rats. *Acta Odontologica Scand.* 37: 377-384. (Cited in U.S. EPA, 1980)

NRC (National Research Council). 1978. Zinc. University Park Press, Baltimore MD. p. 258-261.

Porter, K.G., D. McMaster, M.E. Elmes and A.H.G. Love. 1977. Anemia and low serum-copper during zinc therapy. *Lancet*. p. 744.

Prasad, A.S., G.J. Brewer, E.B. Schoomaker and P. Rabbani. 1978. Hypocupremia induced by zinc therapy in adults. *J. Am. Med. Assoc.* 240(20): 2166-2168.

Riviere, M.R., I. Chouroulinkov and M. Guerin. 1959. Testicular tumors in the rat after injection of zinc chloride. *C.r. hebd. Seanc. Acad. Sci., Paris*. 249: 2649-2651. (Fre.) (Eng. trans.)

Stoner, G.D., M.B. Shimkin, M.C. Troxell, T.L. Thompson and L.S. Terry. 1976. Test for carcinogenicity of metallic compounds by the pulmonary tumor response in strain A mice. *Cancer Res.* 36(5): 1744-1747.

Thompson, E.D., J.A. McDermott, T.B. Zerkle, J.A. Skare, B.L.B. Evans and D.B. Cody. 1989. Genotoxicity of zinc in 4 short-term mutagenicity assays. *Mutat. Res.* 223: 267-272.

U.S. EPA. 1980. Ambient Water Quality Criteria for Zinc. Prepared by the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-079.

U.S. EPA. 1984. Health Effects Assessment for Zinc (and Compounds). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC.

U.S. EPA. 1987. Summary Review of the Health Effects Associated with Zinc and Zinc Oxide. Health Issue Assessment. Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA/600/8-87/022F.

U.S. EPA. 1988. Ambient Water Quality Criteria Document Addendum for Zinc. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC.

Wallenius, K., A. Mathur and M. Abdulla. 1979. Effect of different levels of dietary zinc on development of chemically induced oral cancer in rats. *Int. J. Oral Surg.* 8: 56-62.

Walters, M. and F.J.C. Roe. 1965. A study of the effects of zinc and tin administered orally to mice over a prolonged period. *Food Cosmet. Toxicol.* 3: 271-276.

Woo, Y.T., D.Y. Lai, J.C. Arcos and M.F. Argus. 1988. Chemical Induction of Cancer: Structural Bases and Biological Mechanisms. Vol. IIC, Natural, Metal, Fiber and Macromolecular Carcinogens. Academic Press, San Diego. p. 488-489. In preparation Not available at this time

27. Sharma, R.P. & Reddy, R.V. (1987) Toxic Effects of Chemicals on the Immune System,, Chapter 15 in Haley, T.J., & Brendt, W.O., Eds., *Handbook of Toxicology*, Hemisphere, pp. 555-591.

28. Gaworski, C.L., and Sharma, R.P. (1978) The Effects of Heavy Metals on [³H]Thymidine Uptake in Lymphocytes, *Toxicology and Applied Pharmacology* 46:305-313.

APPENDIX B
SAMPLE CALCULATIONS

SAMPLE HEALTH/HAZARD CALCULATIONS

Lead-Acid Smelting

$$R_c = \left[\left(\frac{UT_i}{UT_T} \right) \times M_{air} \times UR \right] + \left[\left(\frac{UT_o}{UT_T} \right) \times M_{air} \times PS \right]$$

where:

UT_i , UT_o = Inhalation and oral uptake ($\mu\text{g/day}$), respectively (from Table 29)

UT_T = Total uptake ($\mu\text{g/day}$)

M_{air} = Mass of substance emitted to air (from Tables 6-22)

UR = OEHHHA's Unit Risk Factor ($\text{risk}/\mu\text{g}/\text{m}^3$) (from Table 29)

PS = OEHHHA's Potency Slope Factor ($\text{risk}/\text{mg}/\text{kg-day}$) (from Table 29)

$$\begin{aligned} R_c(Pb) &= \left[\left(\frac{0.05 \mu\text{g/day}}{0.05 + 9.38 \mu\text{g/day}} \right) \times 0.026 \text{ g Pb/mi} \times 1.2 \times 10^{-5} \text{ risk}/\mu\text{g}/\text{m}^3 \right] \\ &+ \left[\left(\frac{9.38 \mu\text{g/day}}{0.05 + 9.38 \mu\text{g/day}} \right) \times 0.026 \text{ g Pb/mi} \times 0.04 \text{ risk}/\text{mg}/\text{kg-day} \right] \\ &= 1.03 \times 10^{-5} \end{aligned}$$

$$\begin{aligned} R_c(As) &= \left[\left(\frac{0.16 \mu\text{g/day}}{0.16 + 9.38 \mu\text{g/day}} \right) \times 3.32 \times 10^{-7} \text{ g As/mi} \times 3.3 \times 10^{-3} \text{ risk}/\mu\text{g}/\text{m}^3 \right] \\ &+ \left[\left(\frac{9.38 \mu\text{g/day}}{0.16 + 9.38 \mu\text{g/day}} \right) \times 3.32 \times 10^{-7} \text{ g As/mi} \times 0.49 \text{ risk}/\text{mg}/\text{kg-day} \right] \\ &= 1.60 \times 10^{-7} \end{aligned}$$

$$R_c(\text{normalized}) = \frac{(1.03 \times 10^{-5} + 1.60 \times 10^{-7})}{1.05 \times 10^{-5}} = 1$$

$$R_{CT} = \left[\left(\frac{C_{ann}}{REL_i} \right) \times M_{air} \right] + \left[\left(\frac{UT_o}{REL_o} \right) \times M_{air} \right]$$

where:

- C_{ann} = Average annual ambient concentration ($\mu\text{g}/\text{m}^3$) (from Table 29)
 REL_i = Chronic inhalation Reference Exposure Level ($\mu\text{g}/\text{m}^3$) (from Table 29)
 UT_o = Oral uptake (mg/kg-day) (from Table 29)
 REL_o = Oral Reference Exposure Level (mg/kg-day) (from Table 29)
 M_{air} = Mass of substance emitted to air (from Tables 6-22)

$$\begin{aligned}
 R_{cT}(Pb) &= \left[\left(\frac{0.056 \mu\text{g}/\text{m}^3}{1.5 \mu\text{g}/\text{m}^3} \right) \times 0.026 \text{ g Pb}/\text{mi} \right] \\
 &\quad + \left[\left(\frac{\frac{9.38 \mu\text{g}/\text{day} \times 10^{-3} \text{ mg}/\mu\text{g}}{25 \text{ kg}}}{0.00043 \text{ mg}/\text{kg}-\text{day}} \right) \times 0.026 \text{ g Pb}/\text{mi} \right] \\
 &= 2.36 \times 10^{-4}
 \end{aligned}$$

$$\begin{aligned}
 R_{cT}(As) &= \left[\left(\frac{0.061 \mu\text{g}/\text{m}^3}{0.5 \mu\text{g}/\text{m}^3} \right) \times 3.32 \times 10^{-7} \text{ g As}/\text{mi} \right] \\
 &\quad + \left[\left(\frac{\frac{9.38 \mu\text{g}/\text{day} \times 10^{-3} \text{ mg}/\mu\text{g}}{70 \text{ kg}}}{0.001 \text{ mg}/\text{kg}-\text{day}} \right) \times 3.65 \times 10^{-7} \text{ g As}/\text{mi} \right] \\
 &= 8.48 \times 10^{-8}
 \end{aligned}$$

$$R_{cT}(\text{normalized}) = \frac{(2.36 \times 10^{-4} + 8.48 \times 10^{-8})}{2.36 \times 10^{-4}} = 1$$

$$R_{AT} = \left(\frac{C_{\max}}{REL_i} \right) \times M_{air}$$

where:

C_{\max} = 1-hr maximum ambient concentration ($\mu\text{g}/\text{m}^3$) (from Table 29)

REL_i = Acute Inhalation Reference Exposure Level ($\mu\text{g}/\text{m}^3$) (from Table 29)

M_{air} = Mass of substance emitted to air (g/mile) (from Tables 6-22)

$$R_{AT}(As) = \left(\frac{0.07 \mu\text{g}/\text{m}^3}{0.39 \mu\text{g}/\text{m}^3} \right) \times 3.32 \times 10^{-7} \text{ gAs}/\text{mi} = 5.95 \times 10^{-8}$$

$$R_{AT}(\text{normalized}) = \frac{5.95 \times 10^{-8}}{5.95 \times 10^{-8}} = 1$$

$$R_L = \left(\frac{M_{land}}{TTLC} \right)$$

where:

M_{land} = Mass of substance discharge to land (from Tables 6-22)

TTLC = California Total Threshold Limit Concentration (mg/kg) (from Table 29)

$$R_L(Pb) = \left(\frac{0.68 \text{ gPb}/\text{mi}}{1,000 \text{ mg}/\text{kg}} \right) = 6.81 \times 10^{-4}$$

$$R_L(Sb) = \left(\frac{0.45 \text{ gSb}/\text{mi}}{500 \text{ mg}/\text{kg}} \right) = 9.06 \times 10^{-4}$$

$$R_L(As) = \left(\frac{0.045 \text{ g As/mi}}{500 \text{ mg/kg}} \right) = 9.09 \times 10^{-5}$$

$$R_L(normalized) = \frac{(6.81 \times 10^{-4} + 9.06 \times 10^{-4} + 19.09 \times 10^{-5})}{1.68 \times 10^{-3}} = 1$$

$$R_F = \sum (F_F \times M_{const}) \times S_F$$

where:

F_F = NFPA Flammability Factor (from Table 29)

M_{const} = Mass of constituent contained within the battery

S_F = Qualitative flammability score for each battery (from Table 27)

$$R_F(plastic) = (3 \times 0.91 \text{ g/mi}) = 2.73$$

$$R_F(lead-acid) = 2.73 \times 2 = 5.55$$

$$R_F(normalized) = \frac{5.55}{19.64} = 0.282$$

$$R_R = \sum (F_R \times M_{const}) \times S_R$$

where:

F_R = NFPA Reactivity Factor (from Table 29)

M_{const} = Mass of each constituent contained with in the battery

S_R = Qualitative reactivity score for each battery (from Table 28)

$$R_R(Sb) = (1 \times 0.45) = 0.45$$

$$R_R(As) = (1 \times 0.05) = 0.05$$

$$R_R(Sn) = (1 \times 0.05) = 0.05$$

$$R_R(H_2SO_4/H_2O) = (2 \times 5.09) = 10.18$$

$$R_R(lead-acid) = (0.45 + 0.05 + 0.05 + 10.10 \times 3) = 32.18$$

$$R_R(normalized) = \frac{32.18}{32.18} = 1$$

